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Pain is Mechanism

by
Simon Peter van Rysewyk

School of Philosophy
University of Tasmania

Submitted in fulfilment of the requirements for the Degree of Doctor of Philosophy,
University of Tasmania, March, 2013

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The following publications are cited in this project:

van Rysewyk, S. (in press). Age-differences in face perception: a review of N170 event-related potential studies. In: A. Freitas-Magalhães (Ed.) *Emotional Expression: The Brain and the Face* (V. IV, Second Series). Oporto: University of Fernando Pessoa Press.

van Rysewyk, S. (2011). Beyond faces: The relevance of Moebius Syndrome to emotion recognition and empathy. In: A. Freitas-Magalhães (Ed.), *'Emotional Expression: The Brain and the Face'* (V. III, Second Series) (pp. 75-97). Oporto: University of Fernando Pessoa Press.

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List of Abbreviations

ACC	anterior cingulate cortex
ACH	acetylcholine
AINS	anterior insula
AM	amygdala
ANS	autonomic nervous system
AST	anterior spinothalamic tract
CAS	complex adaptive systems
CGRP	calcitonin gene-related peptide
CIP	congenital insensitivity to pain
CNS	central nervous system
CORT	cortisol
CRH	corticotropin-releasing hormone
CRH-1	corticotropin-releasing hormone receptor 1
CRH-2	corticotropin-releasing hormone receptor 2
DLF	dorsolateral funiculus
DNB	dorsal noradrenergic bundle
E	epinephrine
EM	eliminative materialism
fMRI	functional magnetic resonance imaging
FP	folk psychology
HPA	hypothalamo-pituitary-adrenocortical axis
HPG	hypothalamo-pituitary-gonadal axis
HPP	hippocampus
HPVN	hypothalamic periventricular nucleus

HYP	hypothalamus
IL	intralaminar thalamus
INS	insula
LC	locus caeruleus
LCN	locus caeruleus noradrenergic system
LST	lateral spino-thalamic tract
M1	primary motor cortex
M3	rostral cingulate cortex
M4	caudal cingulate motor cortex
NA	nucleus ambiguus
NE	norepinehrine
NEIM	nervous-endocrine-immune mechanism
NKA	neurokinin A
NO	nitric oxide
NRM	nucleus raphe magnus
PAG	periaqueductal gray
PCG	post-central gyrus
PET	positron emission tomography
PF-CM	parafasciculus (PF) and centromedian (CM) mechanism
PINS	posterior insula
rCBF	regional cerebral blood flow
RGC	nucleus reticularis gigantocellularis
S1	primary somatosensory cortex
S2	secondary somatosensory cortex
SAM	sympatho-adreno-medullary axis

SC	superior colliculus
SIA	stress-induced analgesia
SMA	supplementary motor area
SNS	sympathetic nervous system
SP	substance P
THA	thalamus
TT	theory-theory
VPA	ventral premotor area
VPL	ventroposterolateral nucleus
VPM	ventroposteromedial nucleus

Acknowledgements

The overall aim of this project is to best explain the mind-problem by making the case for a type identity theory of pain. I wish to acknowledge the following people, without whom this project would have been impossible. Most importantly, I thank my wife, Shanna, for her unfailing dedication and understanding, in good times and bad, throughout the course of my PhD. I dedicate this project to her and our adorable children, Adina and Eli. 我非常愛你們。

I wish to thank the University of Tasmania for its financial support of my PhD (Tasmania Graduate Research Scholarship). I extend exceptional praise and attention to Professor Peter Frappell (former Dean of Graduate Research, University of Tasmania) for arranging to finance my PhD following expiration of my Tasmania Graduate Research Scholarship extension in August, 2012.

I wish to warmly acknowledge Dr. Richard Corry (School of Philosophy, University of Tasmania), my primary supervisor, Professor Kenneth D Craig (Department of Psychology, University of British Columbia, Canada) and Assistant Professor Liesbeth Goubert (Department of Health Psychology, Ghent University, Belgium), my co-supervisors, for their expert guidance in this project. I express special gratitude to Dr. Corry for agreeing to supervise me early in 2012, and his exceptional open-mindedness and flexibility. Thanks, Richard.

I thank Professor Jeffrey Summers, Doctor Hakuei Fujiyama, and Doctor Mark Hinder for supervision and assistance during my enrolment in the School of Psychology, University of Tasmania. It is unfortunate our pilot experiments on pain facial expression did not replicate previously reported pain face effects. Finally, I thank Dr. Trung T. Ngo (School of Psychology & Psychiatry, Monash University; Caulfield Pain Management & Research Centre, Caulfield Hospital; Monash Alfred Psychiatry Research Centre) for indispensable

general advice on how to navigate the complexities of scholarship and research.

The following individuals privately supported my PhD financially: Ching-Yi Huang, Tony van Rysewyk, Ben van Rysewyk, Karen Muntz, Lindy Gullett, Kate Reilly, Adrian Bye, Brita Hansen, Shu-Juan Huang, Bernadette van Rysewyk, Genevieve van Rysewyk, Naill Hamilton, Jai Ranganathan, and Jarrett Byrnes. I thank you all.

Finally, I thank Peter van Rysewyk and David van Rysewyk for their encouragement and goodwill.

Thesis Abstract

The mind-body problem is the problem: what is the relationship between mind and body? In this project, I claim that the relationship between the experience of pain and specific physiological mechanisms is best understood as one of *type identity*. Specifically, the personal experience of pain is an allostatic stress mechanism comprised of interdependent nervous, endocrine and immune operations.

In Chapter One, I provide five reasons to prefer type identity theory of mind to dualistic philosophies of mind: it has greater explanatory power; it is more respectful of philosophical and folk intuitions about the causal powers of qualia; it is simpler; it is supported by the causal closure of the physical; and it is continuous with the natural sciences and not separate from them. I describe and challenge four philosophical objections to type identity theory of mind: mental states are excluded; mental states disappear; inverted qualia; and Saul Kripke's claim that type identity theory is false because two individuals could have the same mental state while having different physiological states.

In Chapter Two, I advance a type identity theory of pain supported by a robust theoretical schema as the best description of the mind-body puzzle: *what* is pain? I frame a well established multilevel descriptive view of the physiological mechanisms that describe pain qualia within the context of advancing theoretical descriptions of the nervous, endocrine and immune systems and their functional interdependencies, and descriptions of allostasis, homeostasis, stress and wounds, all constrained in turn by complex adaptive systems theory. A biological individual is a complex adaptive system coping with a physical and social environment, but possessing nested subsystems. Taken together, these descriptions show how pain qualia are type identified with specific neurophysiological mechanisms; namely: (1) somatosensory qualia of pain, including submodality, intensity, duration and location, are best described as the operations of multisubsystem mechanisms in the neospinothalamic tract;

(2) negative emotional pain qualia are the operations of multisubsystem mechanisms in the paleospinothalamic tract; (3) cognitive pain qualia (pain anticipation) are the operations of primary somatosensory cortex (neospinothalamic tract); (4) pain suppression (stress-induced analgesia) are the operations the dorsolateral funiculus pathway and opiate systems (paleospinothalamic tract). The simplest and most parsimonious metaphysical description of these robust relationships is that pain *is* mechanism.

In Chapter Three, I advance a novel polyvagal-type identity theory of pain facial expression to best explain the *explanatory* mind-body puzzle: *how* can pain exist? According to some philosophers, assuming neuroscience explains with what mechanistic operations being aware of a burning arm pain is type identical, it is still impossible for any neuroscientific theory to explain how a specific pain must be correlated with a specific mechanism, as opposed to a different mechanism. Thus, there appears to be an explanatory gap. In this chapter, I will attempt to bridge the explanatory gap in two ways. First, based on the theoretical approach for a type identity theory of pain offered in Chapter Two, I offer a polyvagal-type identity theory of the mammalian pain face. I claim type identity theory of mind best explains how the gap can be bridged. Type identity theory of mind makes a realist assumption that pain is causally responsible for behaviours such as facial pain grimaces and screaming. Second, I attempt to bridge the gap by arguing that the supposed gap assumes that type identity theory must reconstruct type pain identities as formal derivations from laws of nature. Based on actual scientific practice and philosophical considerations concerning explanatory levels, I will show that this is a false assumption. Type identity statements that successfully emerge from mechanistic pain explanation are between different delineations of pain phenomena at the *same* explanatory level; they are intralevel. Although the explanatory gap puzzle correctly shows our incomplete understanding of how pain might be explained by

mechanism, the gap merely asserts a practical limit on our present explanatory successes, and is not in principle unbridgeable on *a priori* grounds, as some philosophers claim.

Eliminative materialists also assert that there is nothing more to pain than mechanism, but deny that pain can be type identified with neurophysiological mechanism. The reason is that pain does not really exist. Eliminativists propose that pain is part of folk psychology which consists of false generalizations ('wounding is the cause of pain') and false theoretical claims ('pain always hurts'). Thus, pain should be eliminated and replaced by an accurate neuroscientific successor theory. In Chapter Four, I assess philosophical arguments and data for and against eliminative materialism of pain. If eliminativism is correct, then the version of type identity theory of pain I propose in this project is strictly false. While pain folk psychology has stimulated much psychological research resulting in improved clinical outcomes for some pain patients, our own intellectual history reveals that any theory can seem successful even when it is radically false. Neuroscience already shows that many folk pain claims are false, while the truth of many others is not known. Eliminative materialism implies the disquieting consequence that the limit of what can be scientifically eliminated or reduced is much closer than we may conventionally think. Alternately, since intellectual history also offers modest evidence of theoretical co-existence between type identity theory of mind and folk psychology, radical theory change as advocated by eliminativism is just one end-point on a continuum of many possibilities. Still, accommodation such as this cannot change the sobering insight that all human knowledge is ultimately provisional. This realization encourages guarded humility about the ontological status of existing folk and scientific pain theories, including the type identity theory of pain, while it fosters an equally guarded optimism concerning our future theoretical prospects.

Keywords: allostasis, biological individual, causal closure of the physical, complex

adaptive systems, dualism, eliminative materialism, explanatory mind-body gap, folk psychology, homeostasis, inverted qualia, mechanism, mechanistic description, mechanistic explanation, metaphysical mind-body gap, pain, pain facial expression, qualia, reductionism, stress, type identity theory of mind, wounds

1 Type Identity Theory of Mind

Abstract

In this chapter, I set the stage for the type identity theories given in Chapter Two and Chapter Three by assessing type identity theory of mind. There are five reasons to prefer type identity theory of mind to dualism: it has greater explanatory power; it is more respectful of philosophical and folk intuitions about the causal powers of qualia; it is simpler, it is supported by causal closure of the physical; and it is continuous with natural science. Using pain as the target mental state, I describe and challenge four philosophical objections to type identity theory of mind: the experience of pain is excluded; the experience of pain disappears; qualia inversion; and Saul Kripke's claim that type identity is false because it is possible that two individuals could have the same mental state while having different neurophysiological states.

Keywords: causal closure of the physical, dualism, folk psychology, inference to the best explanation, naturalism, neuroscience, pain, qualia, Saul Kripke, simplicity, qualia inversion, type identity theory of mind

1 Introduction

The mind-body problem in philosophy of mind is the problem: what is the relationship between mind and body? In this project, I claim that the relationship between the experience of pain and specific physiological mechanisms is one of *type identity*. That is, *being a pain* is type identical with *being the operation of specific neurophysiological mechanisms*. This metaphysical claim concerning the nature of pain is a type identity theory of mind. Type identity theory of mind asserts that mental states are type identical to neurophysiological states (e.g., Armstrong, 1962, 1968; Churchland, 1989; Feigl, 1958; Hill, 1991; Papineau, 2002; Place, 1956; Polger, 2011; Smart, 1959).

At the outset, we need to separate two versions of the identity theory: token identity theory and type identity theory. Token identity proposes that specific mental states, or *tokens* of mental states, are identical with specific neurophysiological states, or *tokens* of neurophysiological states. For example, the pain I currently feel in my left thigh is identical with a neurophysiological state, likely one comprising nervous, endocrine and immune operations. The other version of the identity theory asserts that *states* of specific mental *types* are identical with neurophysiological *states* of neurophysiological *types*. So, in addition to implying that my current pain is identical with a specific state in my body, it implies that the state *being a pain* is identical with a type of neurophysiological state, likely the state *being a specific type of nervous, endocrine and immune operation* (e.g., Chapman et al. 2008).

The identity theory thesis that a mental state is identical with a neurophysiological state entails that all specific tokens of the neurophysiological state are identical with tokens of the mental state. The entailment, however, does not hold for the converse view. This means that assertions of *type identity* are logically stronger than assertions of *token identity*, and have accordingly greater explanatory potential to offer an understanding of the mind and brain (Churchland, 2002; Feigl, 1958; Hill, 1991; Papineau, 2002; Place, 1956; Smart, 1959).

An assertion of type identity implies Leibniz's Law of the Indiscernibility of Identicals; namely, if, *x* is identical to *y*, then for every property *F*, object *x* has *F* if and only if object *y* has *F*. Or, in notation of symbolic logic: $x = y \rightarrow \forall F(Fx \leftrightarrow Fy)$. The converse of the Principle, $\forall F(Fx \leftrightarrow Fy) \rightarrow x = y$, is called the Identity of Indiscernibles; namely, if, for every property *F*, object *x* has *F* if and only if object *y* has *F*, then *x* is identical to *y*. In logic and metaphysics, the conjunction of both principles is usually called Leibniz's Law (Leibniz & Loemker, 1969). In part due to greater explanatory potential, and in part also because assertions of type identity are thought by philosophers of mind to be more controversial than assertions of token identity, type views have been more discussed than token views (Smart, 2007). In line with this trend, the identity theory of pain I advance and defend in this project is type identity.

Some philosophers propose that though pains are neurophysiological operations they still have fundamentally nonphysical, psychical properties, usually termed *qualia*. Philosophers who endorse this philosophy of mind are *property dualists* (e.g., Chalmers, 1996; Descartes, 1637, 1664; Jackson, 1982). If mind and body are fundamentally different kinds of property, according to property dualism, then there is the puzzle of how they are related. According to epiphenomenalism, mental states are caused by physical events, but have no causal influence on the physical (e.g., Jackson, 1981, 1985). In this project, I shall take the type identity theory of pain as denying the existence of such irreducible nonphysical properties. Thus, I reject property dualism and epiphenomenalism. Table 1 compares property dualism with six core philosophical theses of type identity theory of mind. In sections 4.1 and 4.3 of this chapter, I criticize arguments given in support of property dualism.

2 Type Identity Theory of Mind and Qualia

Discussions of type identity in contemporary philosophy of mind typically assume the

idea of a *qualitative state* or a *quale* (Smart, 2007). There is no universally accepted definition of this idea, but there is wide agreement that, following folk psychology, *qualia* (singular *quale*) designate the intrinsic, introspectible states of conscious bodily experiences, such as *being a pain*, the intrinsic, introspectible states of emotions, such as the *feeling of joy*, and the states of sense experiences that we refer to by expressions such as 'tastes minty,' 'smells sulphurous,' 'looks red' (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Kanai & Tsuchiya, 2012; Jackson 1982; Ray et al. 2013; Searle 1992; Tye, 2000, 2006). It is usually stated by property dualists that a mental experience is qualitative if there is something it is *like* to have that experience, and the way it appears to have one of these experience *is* what it is like to have it (e.g., Chalmers, 1996; Jackson, 1982; Ray et al. 2013; Siewert, 2006; Tye, 2000).

To briefly illustrate the idea of qualia, imagine feeling pain, then pleasure following cessation of the pain. What it's like to feel pain is not the same as what it is like to feel pleasure following pain cessation. Thus, the experience of the pain has a quale that the experience of the pleasure lacks. In the same way, the experience of the pleasure has a quale that the experience of the pain lacks. If you have a pain in your head and a cramp in your leg, both stimuli cause in you an experience with a specific qualitative state in virtue of which you can discriminate a pain from a cramp. Your experience of a pain has a qualitative state that your experience of a cramp lacks. An experience of *both* a pain and a cramp presents a pain quale and a cramp quale as opposed to none (e.g., Levine, 2000). Two pains are equal because they both present the same quale of painfulness. Finally, this reasoning applies to cases in which there is no bodily part where painfulness appears to be located. For example, a pain in your left lower leg elicits in you a quale in virtue of which the qualities that characterise the appearance of this part of your body feel painful, even when there is no body part at the place where the pain appears to be located *(phantom pain)*. Thus, the similarity

presented by body parts that appear to feel painful is that the appearances of such parts present the same quale of painfulness.

Since the nature of qualia is subjectivity, two subjects in a psychophysical experiment presented with the same stimulus and showing the same behavioural responses may nonetheless have quite different qualitative experiences. Stimulus-response identity does not entail experiential identity, and such identity cannot be ensured other than by describing the experiential states and how these are experienced. As an example, consider that pain threshold level in psychophysical testing is revealed by means of introspection and then self-report of subjective quality (i.e., qualia). The subjectivity of qualia is consistent with the notion that, in the case of bodily experiences, appearance and reality *coincide*: if I feel pain (i.e., if it appears to me that I am in pain), then I am in pain (there is nothing else that I must satisfy in order for me to be in pain) (e.g., Kripke, 1980). There is an air of paradox when unfelt pains are mentioned. If a pain is not being felt by its subject, then it does not exist.

Most proponents of type identity theory of mind usually limit their philosophical assertions to qualia. The most common version of the type identity theory is the assertion that qualia are identical with specific states that fall within neuroscience. There are several reasons for this limitation. Perhaps the most important of these is that much of the justification for type identity theory (and also much of the motivation), comes from robust evidence correlating somatosensory, emotional and cognitive qualia to neurophysiological states. In this project, I will attempt to type identify pain qualia with complex and interdependent operations in nervous, endocrine and immune systems that describe in concert a single mechanism, the *nervous-endocrine-immune mechanism* (NEIM) (Chapman et al. 2008; Chapman, 2010; Melzack, 1999).

Traditionally, type identity theorists of mind have doubted that there are similar patterns linking such representational states as beliefs and anticipations to specific cross-

system physiological operations. The reason for the denial is that representational (or intentional) states have states that link individuals to phenomena in the *external world* (Hill, 1991; Wickforss 2007). For example, in order to believe that Salman Rushdie is an author, it appears that an individual must have a concept that he uses to represent information about Salman Rushdie. By contrast, it is usually thought that qualia are purely internal. However, in Chapter Two, section 5.6, I present sufficient experimental evidence to support the description that the somatotopic organization of primary somatosensory cortex (S1) best describes the anticipation of pain in a specific body part.

In the next section, I describe five reasons to prefer type identity theory of mind to dualism.

3 Five Reasons to Endorse Type Identity Theory of Mind

3.1 Greater Descriptive and Explanatory Power

The first reason to prefer type identity theory compared to dualism is that type identity possesses greater *explanatory power* (Place, 1956). Type identity theory observes the robust empirical correlations between mental states and specific neurophysiological states and between mental states and specific behavioural states (e.g., facial expressions) reported in the neurosciences. Thus, when a functional neuroimaging study strongly correlates a cognitive operation with a brain region (e.g., perceiving human faces with cortical activity in the fusiform face area (FFA); van Rysewyk (2010), for review), or when an event-related potential[1] (ERP) study identifies a cognitive operation with a change in electrical potentials (e.g., age-differences in human face perception correlated with a N170 ERP, van Rysewyk, (in press), for review), the type identity theory claims that the neural operation *is* the

[1] An event-related potential (ERP) is a reliable electrophysiological brain response following a specific sensory, cognitive or motor stimulus (i.e., an 'event'). ERPs are measured with electroencephalography (EEG) or with magnetoencephalography (MEG) and have clinical and research applications.

cognitive operation: increased activity in the FFA *is* the visual perception of human faces; increased activity in the N170 *is* the age-difference in human face perception.

Note that there are many possible reasons why neurophysiological states and mental states such as pain might be empirically correlated: (1) the neurophysiological operations are a condition for pain; (2) the neurophysiological operations are part of the cause of pain; (3) the neurophysiological operations are part of the effects of pain; (4) the neurophysiological operations corresponds only indirectly to pain; (5) the physiological operations are what pain can be *type identified* with (i.e., the *type identificand*) (e.g., Churchland, 1989, 2002; Smart, 1959).

A descriptive and explanatory account of pain requires the *type identification* of some class of neurophysiological operations with pain; that is, the type identity theorist wishes the correlational data to both describe and justify interpretation (5). Interpretation (5) has the form, 'an instance of *X* occurs only when an instance *Y* occurs' (Hill, 1991; Smart, 1959). Type identity claims of the form '*X* is the same thing as *Y*' are then derived from propositions specifying interpretation (5). Propositions specifying type identities are more fundamental than propositions specifying correlations because only the former can bring a chain of explanations to an end (Churchland, 1989; 2002; Hill, 1991; Smart, 1959). Correlations by definition do not exclude all alternatives except interpretation (5). However, the reliable correlation of a neurophysiological operation *x* with pain *is* informative because this correlation may be the right path for describing and explaining the *type identificand.* Correspondingly, a neural event *z* that does not reliably correlate with pain indicates that this path may not be the one (Churchland, 1989; Frith et al. 1999).

Type identity theory asserts that it offers the *best description* and the *best explanation* for these robust correlations. That is, the best way of describing and explaining the robust correlation between pain qualia and specific neurophysiological states is to assert that pain is

type identical with those states. According to a kind of abductive inference called *Inference to the Best Explanation*, it is appropriate to prefer descriptive theories that offer the *best explanations* of phenomena in their domains, all other things being equal. Thus, if a theory provides the best explanation of all the data that are relevant to pain, then we are allowed to believe that descriptive theory, relative to the alternatives (e.g., dualism). Since type identities are thought by type identity theorists to satisfy this stipulation much better than dualism, type identity philosophers infer that type identity theory *best describes* the mind (Hill, 1991; McLaughlin, 2010; Place, 1956; Smart, 1959).

To illustrate inference to the best explanation, consider the puzzle whether nonhuman animals can experience pain in the sense of pain qualia, as characterized above. To consider nonhuman animal pain is to reflect on the possibility that, in Nagel's (1974) phrase, there might be 'something it is *like*' to be an animal in pain. Nagel disputes our capacity to know, conceive, or describe in scientific terms *what* it is like to be a (e.g.) bat in pain, but he assumes that there *is* something it is like. However, it appears that Nagel's skeptical view overlooks the crucial difference between believing with justification *that* an animal is in pain and knowing *what* it is like to be an animal in pain. The difference is crucial because the personal experience of a pain implies a negative conclusion only about the latter. To imply a negative conclusion about the former we must also assume that pain has no measurable effects on behaviour; that is, we must assume epiphenomenalism (e.g., Jackson, 1982, 1985). But since I reject epiphenomenalism, and make the additional assumption that pain *does* have effects on behaviour, as I discuss in the next section and in Chapter Three, then inference to the best explanation may be used to enable its description.

As a research strategy, inference to the best explanation justifies the description of conscious pain in animals based on the robust explanatory and predictive power that is gained from such descriptions. Without such descriptions, we would be unable to make sense of (e.g.)

facial displays of pain in nonhuman mammals (Keating et al. 2012; Langford et al. 2010; Leach et al. 2012; Sotocinal et al. 2011). This approach depends on conventional scientific reasoning: of two hypotheses (e.g., 'pain does have effects on facial behaviour', or 'pain does not have effects on facial behaviour'), the one that better explains the phenomenon of pain, and is consilient with well established science, is the one to be preferred. An aim of this project is to show that type identity theory of mind is more successful in showing that conscious pain *better describes* and *better explains* the observed pain behaviour, compared to dualism.

One way to support the strategy of inference to the best explanation in a type identity theory of pain is to fortify it with an empirically informed understanding of the *relationship* between neurophysiological mechanism and pain qualia. If it was known with what mechanisms pain is correlated, then this knowledge could be used to infer its presence and type identity in cases where that correlate is satisfied, so long as other kinds of explanations can be shown to be less satisfactory (e.g., dualistic or epiphenomenal explanations). If pain qualia are absolutely epiphenomenal, then a search for the correlates and succeeding type identities of pain will fail. On the empirically supported assumption that pain is an evolved feature of human beings and most nonhuman mammals, including such species as birds (e.g., Machin, 2005), fish (e.g., Rose et al. 2012; Sneddon, 2012), possibly crabs (e.g., Elwood & Appel, 2009; Magee & Elwood, 2013) and prawns (Barr et al. 2008), and therefore that epiphenomenalism is *false*, then an attempt to understand the neurophysiological correlates of pain may offer the best opportunity of type identifying it. This is a reason why the type identity theory of pain offered in this project is situated within the *descriptive context* of advancing theories of the nervous, endocrine and immune systems, and theories of allostasis, homeostasis, stress and wounds, all constrained in turn by complex adaptive systems theory.

These considerations uncover an interesting methodological question that I will very

briefly consider; namely, how do you know what you are looking at? Pain researchers and type identity theorists assert that there is a class of neurophysiological operations correlated *always* and *only* with pain, and that such activity is type identifiable as pain. Assuming there is such a class of neurophysiological operations, knowing that *these* measured operations belong to pain may be found only very indirectly. It is quite possible that a pain researcher may be looking at an instance of the class without even comprehending that it is an instance. This will occur if, as is likely, a component part of pain does not possess a defining property (the *type identificand*) that is conspicuous to the researcher, but is comprehensible only through the presence of a more complete *theory* of mechanism (Churchland, 1989; Frith et al. 1999).

This problem can be illustrated as follows: How would a person know, independently of Antoine Lavoisier (1743-1794) and Joseph Priestley's (1733-1804) studies on oxygen, that metabolizing, burning and rusting are the same mechanical operation, but that lightning and sunlight are not? What properties would be salient to look at? And if someone did serendipitously guess that the first three phenomena have a common operation – the *identificand* – how would that notion be tested and evaluated? Even granting an ancient astronaut left a message on the lab bench saying, 'good news: rusting is slow oxidation', no one could know what the message could possibly mean.

This is not to claim that looking for the neurophysiological correlates of pain is not productive. After all, much of the motivation for type identity theory, and also much of the justification, comes from robust correlations linking qualia to neurophysiological operations. Correlational data obtained from the use of neuroimaging scanners in hundreds of studies has advanced scientific understanding of chronic pain, has led to recent attempts to develop better diagnoses and drug treatments, and correlational neurotechnologies may contribute to the development of a 'pain phenotype' that could aid in diagnostic and treatment choices of

chronic pain conditions (Borsook et al. 2010, for review). The immediate point I wish to make is that it is prudent to acknowledge the limitations and to understand that they are not simply technological, but arise also from the need to develop a *philosophical* framework for comprehending mechanism (Churchland, 1989; Frith et al. 1999).

3.2 Respectful of Philosophical Intuitions about Qualia

The second reason to prefer identity theory is that type identity is the only theory that fully respects philosophical intuitions about the causal powers of qualia. For example, we believe that pains are causally responsible for such behaviours as facial grimaces, limb-guarding, crying out, and also for much of our talk and thought about pain. We attribute important causal powers to pain, but neuroscience indicates that these causal powers also reside in the physiological operation that is type identified with pain. Accordingly, if pain is distinct from that physiological operation, we will be required to infer either that pain is *epiphenomenal*, having no causal powers in its own right, but merely appearing to have them because of its relation with the neurophysiological state (Jackson, 1981, 1985). In itself, epiphenomenalism is not an attractive view. It obliges us to believe that mental states, even though they are caused by neurophysiological operations, have *no effects* on the world. This seems a very strange kind of causal power. For example, if pains don't *cause* pain behaviour how can it be that your telling me that you are in pain gives me any reason for supposing you are? Moreover, if pain is absolutely epiphenomenal, then a search for fundamental type identities of pain will fail. In fact, if pain is completely epiphenomenal, then it cannot have evolved by natural selection (James, 1979, 1890).

Some philosophers wish to assert that the very notion of qualia is deeply wrong about the nature of the brain-mind. *Eliminative materialists* propose that the idea of pain qualia as conceived by common-sense radically misdescribes the nature of pain; thus, the claims of

common-sense concerning pain designate nothing that is real (e.g., Churchland, 1981; Churchland, 1989; Dennett, 1978; Hardcastle, 1999). Like philosophical dualists, eliminative materialists argue that pain cannot be type identified with neurophysiological mechanisms. However, unlike dualists, eliminativists claim there is nothing more to pain than what occurs in the brain. The reason pain is not irreducible is not because it is non-neurophysiological; instead, it is because pain (and all mental states), as conceived by everyday folk common-sense, *do not really exist*. For example, philosopher Daniel Dennett (1978) has claimed that our concept of pain is false because it comprises necessary properties like awfulness and infallibility that cannot co-exist in view of a well-known phenomenon he terms *reactive disassociation*. I will assess pain eliminativist materialism in Chapter Four.

3.3 Simpler

Third, type identity sees only *one category* of states where other mind philosophies see two. The view of reality that type identity theory offers is *simpler*, and more *coherent*, than the view that is offered by property dualism, which proposes that qualia are ontologically unique states, having no descriptive and explanatory role in physics, biology, or any other natural science (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Jackson 1982; Ray et al. 2013; Tye, 2000, 2006). Whereas type identity theory sees only a single state – a quale that is a physiological state – property dualism sees two. All other things being equal, I think it is reasonable to say that simpler theories are preferable to complex ones. J. J. C. Smart, an early proponent of identity theory of mind, formulated this principle of ontological parsimony in the following passage:

If it be agreed that there are no cogent arguments which force us into accepting dualism, and if the brain-process theory and dualism are equally consistent with the facts, then the principles of parsimony and simplicity seem to me to decide overwhelmingly in favour of the brain-process theory. (Smart, 1959, p.156)

The first reason appeals to Occam's razor, which cautions that entities are not to be multiplied beyond necessity. Occam's razor is thought by some philosophers to offer a compelling reason to prefer type identity to dualism (e.g., Churchland, 2002; Hill, 1991; Papineau, 2002; Smart, 1959). Is type identity theory simpler? Type identity theory of mind and dualism are similar in claiming that reality comprises a category consisting of mental states and also a category consisting of neurophysiological states. But, dualism asserts that the two categories are together irreducible, whereas type identity asserts that one of the categories is reducible to the other. Is an assertion of irreducibility *simpler* than an assertion of reducibility? This question might miss the point at issue, for ultimately type identity theory asserts only *one* category of states where dualism asserts two. Compared to dualism, type identity better serves human longing for ontological unity.

3.4 Causal Closure of the Physical

In metaphysics, type identifying pain with physiological mechanism is an example of *ontological physicalism,* which is a theory concerning the nature of reality. As a metaphysical claim, physicalism asserts that everything is physical (Stoljar, 2009). Applied to pain, physicalism asserts that pain is physical. As a development of the type identity theory of mind, type physicalism claims that every mental property is type identical with some physical property. Thus, for every actual mental property *F*, there is a physical property *G* such that

F=G. *Type pain physicalism* claims that the property *being a pain* is type identical with the physical property *being the operation of specific neurophysiological mechanisms*. Thus, for every actual pain property *F*, there is a physical property *G* such that *F=G*.

The most influential argument for physicalism in contemporary metaphysics is the causal closure thesis (Stoljar, 2009). According to one version of the causal closure thesis, the Causal Closure of the Physical Argument, every physical effect has an immediate sufficient physical cause (e.g., Papineau, 2009). The thesis comprises three requirements: every physical effect has a cause which is *physical*, *immediate*, and *sufficient*. I will examine these requirements in that order.

The first requirement says that for any physical effect, there will always be a prior *physical* cause. For example, the contraction of muscle fibres in my leg is caused by electrochemical operations in my nerves, which is caused by operations in my motor cortex, and so on. This entire sequence can be explained solely in terms of the explanatory resources offered by physics itself, and without invoking any other subject domain. Since physical effects can be explained without leaving the physical realm itself, physics does seem to be causally closed.

The second requirement that the physical cause be 'immediate' is needed to exclude the possibility of physical causes which produce their physical effects only via nonphysical intermediaries. Consider that every bodily movement is fully determined by some prior brain operation, but that these brain operations only produce the bodily movements *indirectly*, by first producing some unique mental quale, which then affects the nervous system, and so on (e.g., Lowe, 2000). This organization would guarantee that every bodily movement had some (indirect) sufficient physical cause (namely, the prior brain operation), but it wouldn't necessarily guarantee the causal closure of the physical, since it wouldn't require that the direct causes of physical effects must be physical, and therefore wouldn't guarantee that

every physical effect had a prior *physical* history. The requirement that every physical effect have an *immediate* physical cause excludes any mixed physical/mental/physical histories of this type, since it guarantees that any physical effect has an immediately prior physical cause, which in turn has an immediately prior physical cause, and so on.

Finally, the third requirement that the physical cause be 'sufficient' is needed to establish that it causes the physical effect by itself and not solely in virtue of its *conjunction* with some ontologically unique nonphysical cause such as a nonphysical quale (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Jackson 1982; Tye, 2006). Now, suppose that some neural operation is caused by the conjunction of some physiological operation *and* some unique mental cause. Then, that neural operation would have *a* physical cause (the physiological operation), but this cause would not have sufficed on its own in the absence of the unique mental cause. To ensure that the causal closure thesis is philosophically significant, the requirement is that every physical effect has a physical cause that *suffices on its own*.

While the causal closure of the physical lies below the surface in the type identity theory, causal closure plays an essential descriptive and explanatory role in it. If causal closure were not true, then some physical effects would not be determined by prior physical causes at all, but by ontologically unique mental causes such as nonphysical qualia. I challenge the idea that pain qualia are nonphysical in section 4 below. Since the causal closure of the physical is the most influential argument for physicalism in contemporary philosophy (Stoljar, 2009), I will briefly consider three objections to it.

3.5 Three Objections to the Causal Closure of the Physical

3.5.1 Defining the Meaning of 'physical'

The causal closure of the physical presupposes a notion of the *physical* domain which requires clarification (Crane & Mellor, 1990). In response to this call, Papineau (2009) defines *physical property* by reference to the fundamental categories of physical *theory*. Accordingly, if physical theory explains the property of having pain, then having pain is a physical property. Similarly, if physical theory explains the property of being a neuron then it too is a physical property. Stoljar (2009) names this the *theory-based conception of the physical*.

An obvious objection to this conception of the physical is that it is circular: it invokes the idea of something physical (a theoretical explanation) to explain a physical property. Characterizing one type of physical thing by invoking another is circular reasoning; thus, the theory-based conception is not a legitimate description of the nature of the physical. A response to the objection is that circularity is only a problem if the theory-based conception is viewed as offering a *conceptual analysis* of the idea of the physical. When philosophers analyse a concept or idea, they typically attempt to offer a conceptual analysis of the notion in question, i.e. to explain it in *other* terms. Applied to pain physicalism, this view may entail the idea that every pain concept or predicate is described in terms of a physical concept or predicate.

However, there is no reason why the theory-based conception should be viewed as attempting to offer a conceptual analysis. We possess many concepts that we comprehend without knowing how to conceptually analyse (Lewis, 1970). In most instances, explanation owes more to abduction (Leake, 1998) or family-resemblance reasoning (Wittgenstein, 2009) than to conceptual analysis. These types of reasoning both involve knowing how to generalize usefully from observed cases and to draw suitable analogies from the familiar to

the unfamiliar. They are acquired by recognizing a thing or event as relevantly similar to a familiar class and inferring that the thing or event has a cluster of properties similar to that of the class (Leake, 1998; Wittgenstein, 2009). In the day-to-day business of life, and in actual scientific practice, abduction launches powerful predictions that turn out to be correct and environmental and imaginative manipulations that prove to be successful (Churchland, 2011).

It is open to a pain physicalist to analyse pain expressions in *topic-neutral* (but not physical) terms, which means that a physicalist could reject conceptual analysis. Topic-neutral terms are neither mental nor physical, but when conjoined with any theory increase the explanatory power of the theory (Smart, 1959). The terms 'waxing', 'waning', 'going on', 'occurring', and 'intermittent' are topic neutral. So there seems no reason to suppose that the theory-based conception is offering anything else but a *way* of comprehending the idea of the physical (cp. Jackson, 1988). Understanding the physical should not be understood as the demand for a conceptual analysis.

Finally, and to intimate an alternative to the theory-based conception of the physical, *physical* might be equated with *mechanistic*. Contemporary biology discovers and explains by delineating the mechanisms responsible for a phenomenon (e.g., pain). The phrase '*theory* of pain' is not typically used; general knowledge in pain science is represented by diagrams of mechanisms. So, the *physical* might be described as anything that is composed of component parts and component operations that together perform a function (Bechtel & Abrahamsen, 2005). The causal-closure thesis of the physical will then propose that every effect that is mechanically constituted must have a cause that is mechanically constituted. Thus, everything that has mechanically constituted effects must itself be mechanically constituted. I merely table this suggestion due to its seeming plausibility. Mechanism, mechanistic description and explanation are essential to the type identity theory of pain I offer in this thesis, and are considered more fully in Chapters Two and Three.

3.5.2 Hempel's Dilemma

The second objection I will consider claims that the theory-based conception leads to a version of physicalism that is either false or trivial. The original version of this dilemma was described by Hempel (1970) and formulated as follows: if physicalism is defined by way of contemporary physics, then it is false. It is false because contemporary physics is incomplete. Still, if physicalism is defined by way of a future physics, then it is trivial. It is trivial because what a future physics contains is not amenable to accurate prediction. The conclusion of the dilemma is that Papineau's (2009) notion of the physical lacks clarity.

I will only respond to the first horn of the dilemma. The first horn states that current physics is incomplete. Assume this is true. Nonetheless, it may be *rational* to believe that physics is complete, or one day will be (cp. Lewis 1994, Melnyk, 2003). After all, isn't it rational to believe that the most current physics is true? A philosopher might choose to deny this claim; but this is not something that most philosophers are attracted to (Stoljar, 2009). So, as I wish to answer the question in the affirmative, it would then be rational as a special case that the best hope for a description of pain is to follow the sciences. If so, then the domain of pain physicalism (and therefore type identity theory of mind) coincides with that of the sciences. In this case, type identity theory of mind is part of *ontological naturalism* (Papineau, 2002, 2009).

What might be mistaken about this response is that it is still wrong to *define* physicalism with respect to the physics that happens to be true (cf. Laudan, 1981). The reason is that whether a physical theory is true or not relies on the (contingent) facts; but whether a property is physical or not is not reliant on the (contingent) facts. To illustrate, take caloric theory of heat. Caloric theory of heat is false (although it might have been otherwise) and so it is not now reasonable to think it true. However, the essential property of having caloric according to caloric theory – the property of having caloric fluid in the spaces between atoms

– is a physical property, and a counterfactual temperature-increase in our world explained by caloric theory would be a temperature-increase of which physicalism is true. But it is difficult to accept how this reasoning could be correct if physicalism were *explicitly defined* by way of the current physics or by the physics that happens, *as a matter of contingent fact*, to be true in our world.

In current philosophy of mind, physicalism is often presented as much more than a metaphysical claim. Likely *because* of its connection to the sciences, physicalism is sometimes portrayed as a complete package of views. The package of views contains both the metaphysical thesis and methodological naturalism as only *two* parts. Other parts of the package contain items such as the unity of science, empiricism, objectivity, and more (Stoljar, 2009). I see the complete package of views (including the metaphysical thesis and ontological naturalism) simply as *naturalism*. Since most philosophers are not inclined to deny that it is rational to believe most physics is reliable, it seems reasonable to infer that what many physicalists of mind – and therefore type identity theorists – are really concerned with is the naturalistic descriptive project (Stoljar, 2009). Indeed, most physicalists endorse methodological naturalism as a matter of fact and sometimes use the same methods of investigation as the natural sciences (e.g., Bechtel, 2007; Churchland, 1986, 2011; Flanagan, 2009; Papineau, 2002, 2009; Quine, 1960, 1974). I regard this as the fifth reason to prefer type identity theory of mind to dualism.

Concerning the first horn of the dilemma then, ontological naturalism can offer two assertions: one, metaphysics is to be conducted in a way that is continuous with the sciences and not separate from them; two, as a matter of fact, the metaphysics that is suggested by the methods of science is physicalism. I think it is rational to believe both that most current physics is reliable *and* that most *methods* of current physics are reliable. Since it is reasonable to define physicalism with respect to the science that happens to be true, it is misleading to

treat physicalism as a piece of metaphysics as opposed to a piece of science. They are both parts of the naturalistic project. Consequently, it is rational to believe that natural science will explain pain. In Chapter Two, I exploit this reasoning to advance a general descriptive schema for a type identity theory of pain.

3.5.3 Mental Experiences and Physical Effects

Finally, some philosophers object to the idea of causal closure and claim that mental experiences don't have physical effects. They argue that mental states are not causally closed because such states cause *actions* as opposed to physical effects (e.g., Hornsby 1997, Sturgeon 1998). Actions are not part of the subject matter of the physical sciences, and thus not the type of effects ensured to have physical causes by casual closure. Thus, there is no reason to think that the status of mental states as causes of actions is explainable by physics. A difficulty with this objection is that actions aren't the only effects of mental states. Mental states can also cause unambiguously *physical* effects. My desire to avoid a noxious stimulus will typically have precisely that effect. Thus, according to casual closure, the movement of my hand when I withdraw it from the fire has a purely physical cause (Papineau, 2009).

3.5.4 A Descriptive Comparison of Type Identity Theory of Mind and Property Dualism

Table 1 presents the core philosophical theses of type identity theory of mind described above, and their relation to property dualism. In the following section, I will consider four philosophical objections to type identity theory of mind.

Table 1: The Six Core Philosophical Theses of Type Identity Theory of Mind and Their Relation to Property Dualism, Adapted From Polger (2011)

Philosophical Thesis	Type Identity Theory	Property Dualism
Realism – Pain qualia are real.	Yes	Yes
Physicalism – Pain qualia are physiological.	Yes	No
Minimal Reductionism – Pain qualia are nothing more than physiological mechanism.	Yes	No
Type Identity – Pain qualia are type identical to physiological mechanism.	Yes	No
Naturalistic – Philosophies of pain are both metaphysical theories *and* scientific theories.	Yes	No
Theoretical – Metaphysical theories of pain can be assessed according to their theoretical virtues (e.g., simplicity), *and* competing empirical predictions.	Yes	No

4 Four Philosophical Objections to Type identity Theory of Mind

In this section, I will consider four philosophical objections that together form a distinctive challenge to type identity theories of mind and physicalism. To introduce the objections in this section of the chapter[2], I will adapt an idea from philosopher Saul Kripke

[2] I will not consider in this project the 'multiple realization' (MR) objection to identity theories of mind (e.g., Fodor, 1974; Kitcher, 1982; Putnam, 1967). Very briefly, Putnam (1967) proposed that mental states such as pain could be experienced by humans and nonhuman animals like dogs and octopuses. As these animals appear to be physiologically varied, it appears that it is not true that the mental state type pain can be identified with a *single* physiological state type. Even if it was true that animals all share physiological operations when in pain, it is not a law of nature that they *must*. Thus, the type identity theory of mind is false. Identity theorists have focused on two main lines of response to the challenge of MR: (1) What is the extent of physiological variation among animals that we think have mental states similar to our own? Identity theorists have claimed that the evidence for physiological variation is inconclusive, and that neuroscientific practice predicts convergence and constraint instead of multiplicity (e.g., Couch, 2004; Bechtel & Mundale, 1999; Polger, 2009; Shapiro 2004; Zangwill, 1992). Bechtel & Mudale (1999) argue that a reason for overestimating the prevalence of MR may be

(1980). Suppose God created the entirely *physical* universe in purely physical terms. For example, God created such things as the distribution and states of elementary particles throughout space and time, together with the laws governing their behaviour. Now, did God have to do something *more* in order to provide for pain? A property dualist or epiphenomenalist philosopher answering this question in the affirmative implies there is *more* to pain than the purely physiological facts. For example, it entails that pain qualia require *nonphysical properties*; and such properties would not exist in a purely physical world (e.g., Block, 1980; Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Jackson, 1982; McGinn, 1991; Nagel, 1974; Ray et al. 2013; Searle, 1992). Alternately, type identity theorists and physicalists would answer the question in the negative. They would say that by creating the physical universe in purely physical terms, God thereby determined *all* the mental facts about the organisms, including physiological facts about pain. According to type identity theory of mind, there is no *metaphysical mind-body gap* between the facts of mental experience and the facts of mechanism.

Some philosophers and scientists think that the following four objections to type identity theory of mind are at least conceivable, and some that they are possible (e.g., Chalmers, 1996, 1999; Derbyshire & Raja, 2011; Feinberg, 2012; Jackson, 1982; Ray et al. 2013; Searle, 1992). These thinkers have argued that if the objections are so much as a bare *possibility*, then type identity theory is false and some kind of dualism of mind is true. Recall that the version of type identity theory asserted in this project is the view that pain is type identical to specific neurophysiological mechanisms. This implies that true factual statements about pain experiences or qualia are alternative ways of talking about those same

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that the mental and physiological types were not individuated at *matching granularities*: If we individuate mental states *coarsely* and physiological states *finely*, then it may appear that more than one physiological state type matches a single mental type and therefore that MR is true; (2) What types of physiological variation is required for MR? How much is required? It appears that not every difference between two animals is a case of or evidence of MR. Some mental differences count as differences in mental type, and some do not. Some physiological differences are important to mental categorization, and some are not (e.g., Polger 2009; Shapiro 2000, 2004).

physiological operations; that is, redescriptions of them. But, if pain qualia are *possibly* nonphysical, then truths about pain qualia are *not* redescriptions of specific physiological facts, and type identity theory is false.

A key question concerning the objections below is how we should understand 'conceivable' and 'possible'. The objections are all unanimous in proposing that if nonphysical pain qualia are *conceivable,* then they are *possible.* I will take *conceivability* here in a broad sense, where conceiving is something like "the capacity that enables us to represent scenarios to ourselves using words or concepts or sensory images, scenarios that purport to involve actual or non-actual things in actual or non-actual configurations" (Gendler & Hawthorne, 2002; Yablo, 2002). These scenarios must be conceived such that arbitrary details can be included without creating any logical contradiction (Chalmers, 1999). Possibility in this context is generally understood as *metaphysical possibility*, where a statement is metaphysically possible if and only if it describes a way things might have been without implying contradiction (Gendler & Hawthorne, 2002). This sense of possible also indicates one plausible sense for the phrase 'type identical'; that is, if the idea of nonphysical qualia implies a logical contradiction, then pain is type identical to the physiological facts. Taken another way, if nonphysical qualia are possible, pain is not in that sense type identical to the physiological facts, and type identity theory of mind is false. If that is correct, then to prove that nonphysical qualia are possible would be to disprove type identity theory of mind.

4.1 The Exclusion of the Experience of Pain

The first philosophical objection I will consider is that any type identity theory of pain will always *exclude* the qualia of pain: that is, the feeling of pain as aversive or threatening, as intense, sharp, dull, or chronic, and so on (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Nagel, 1974; Ray et al. 2013). The objection implies that type identity theory

can never really explain pain qualia because it cannot show *what it is like* to feel pain if you have never felt it. Even if I had an extraordinarily rare pain phenotype such as congenital insensitivity to pain (CIP), thus being entirely ignorant of painfulness in myself, learning the type identity of the tetrodotoxin-sensitive voltage-gated sodium channel gene *SCN9A* with painfulness (Cox et al. 2006; Cox et al. 2010) doesn't tell me *what it is like* to feel pain. Understanding this type identity claim cannot inform me how to introspect a specific pain quale, or how to discriminate a pain quale from a cramp quale. So, the type identity description 'pain = the operation of the gene SCN9A' necessarily excludes something essential to pain; namely, the personal experience of pain itself. It seems that the personal experience of pain is forever beyond type identity theory of mind.

The point concerning discriminating pains may be ceded to the objection. In fact, it is entirely true. To possess the perceptual capacity of discriminating pains demands more than *knowledge* of the identity of *SCN9A* with pain: it demands that the type identity also be *true of* oneself. It demands that *a person actually has a functioning instance of SCN9A that the identity claim specifies*. A person with CIP has a dysfunctional *SCN9A*; and so he or she will remain entirely ignorant where pain qualia are concerned. Knowing the type identity claim specifying that gene will be utterly unhelpful in that regard.

However, this doesn't imply any inadequacy of the type identity description, especially since that identity is framed by an accurate explanatory reduction of what causes CIP at the *micro* level (namely, the nonsense mutation of $Na_v1.7$, the α-subunit of *SCN9A* that is expressed at high levels in peripheral sensory neurons, mostly in nociceptive small-diameter dorsal root ganglia neurons), and how to treat that disorder and closely related hereditary pain disorders such as Paroxysmal Extreme Pain Disorder (PEPD) and Primary Erythermalgia (PE) (namely, target the expression of $Na_v1.7$ in *SCN9A*).

This objection appears to rest on two mistakes. The first mistake involves the misconceived expectation that *having* a specific perceptual capacity (being able to have and discriminate a pain quale) necessarily results from *knowing* a specific type identity (i.e., 'pain = the operation of the gene SCN9A'). The two are not equivalent; and simply knowing the identity will not provide a person with the capacity. However, *if the type identity is true of you* – if you have the functioning gene – then you will have the capacity at issue. You will be able to identify and discriminate a pain by immediate personal reaction to the presented pain quale.

The second mistake appears to involve assuming that if the personal experience of pain is accurately explained by a type identity claim, then a person who understands that identity will be *caused* to feel pain. The following analogies illustrate the mistake in this expectation. If I genuinely understand the nature of visible light, will I thereby see light? If I understand the nature of pregnancy, will I thereby become pregnant? If I understand the nature of physical wounding, will I thereby become physically wounded? In all analogies, the answer appears negative. To feel pain, a specific range of complex physiological operations have to occur, interdependently, as I will show in detail in Chapter Two. To embody pain in the human face, the brain-heart-face mechanism (Porges, 2001, 2006) must be active (Chapter Three). Merely *understanding* that the NEIM must be operant will not itself activate the NEIM. Thus, the first objection asks too much of type identity theory if it is asked to *cause* its target phenomenon; namely, an experience of pain, simply by understanding the theory.

4.2 The Experience of Pain Disappears

This philosophical objection asserts that type identity theory concerning pain makes pain disappear. Broadly, the objection is that if type identity theory identifies pain with a

specific mechanism, then pain itself either is not real or disappears (e.g., Chalmers, 1996; Ray et al. 2013; Searle, 1992). Using this conception of 'type identity', it was then reasoned that because it is observably obvious that a pain quale is real, it cannot be reduced to neuroscience. This misunderstanding trades on an idiosyncratic view of type identity, where it is expected that in science, type identity claims make mental experiences disappear. I think this expectation is misconceived. Temperature was ontologically reduced to mean molecular kinetic energy, but no person expects that temperature therefore ceased to be real or became scientifically disrespectable or redundant. Visible light was ontologically reduced to electromagnetic radiation, but light did not disappear. Instead, scientists understand more about the real nature of light than they did before 1873. Light is real, no doubt; and so is temperature. Some expectations about the nature of temperature and light did change, and scientific progress does occasionally require rethinking what was believed about phenomenon. In certain instances, previously respectable states and substances sometimes did prove to be unreal. The caloric theory of heat did not survive rigorous experimental testing; caloric fluid thus proved to be unreal. Thus, the type identity of pain with the NEIM means only that there is a *description* and an *explanation* of the phenomenon. Scientific descriptions and explanations of phenomenon do not typically make them disappear (Churchland, 1993).

4.3 Inverted Qualia

Many philosophers and some scientists have objected that the facts of any mental experience are necessarily *underdetermined* by *any and all physical* facts, including all physical facts that may become known about that experience. When philosophers say '*p* is underdetermined by *q*', they mean that *p* cannot be formally deduced from *q*; *q* may provide evidence for *p*, but not absolutely conclusive evidence. Thus, it is inferred that facts of mental experience must be distinct and independent facts in their own right, a class of facts that

cannot ever be described and explained in physical terms. Since pain is a mental experience, it follows that pain cannot be described and explained in terms of physiological mechanisms. This conclusion is frequently communicated in the *inverted qualia argument*, which the third philosophical objection I will address in this chapter.

To introduce the argument, consider a scenario in which a single person's qualia are inverted with respect to his qualia at an earlier time. Thus, imagine that you awaken one morning, and begin having rather extraordinary colour experiences: light colours now appear dark, dark colours now appear light. You venture outside with growing interest, and look around. Grass presents a quale which yesterday you called 'magenta', a stop sign looks cyan, and the sky looks yellow. Your ability to discriminate colour stimuli appears completely unaffected: every pair of stimuli you could discriminate yesterday you can discriminate as well today. It is just that every colour you now perceive looks like a colour negative (Wittgenstein 2009, 284).

Now imagine this intrapersonal scenario in an *interpersonal* context: 'it makes sense, or seems to make sense, to suppose that objects we both call green look to me the way objects we both call red look to you' (Block, 1980, 287-288). For example, you and I are both looking at a green car next to a red stop sign. The colour experience of looking at the car is just the one that you have when you look at the stop sign. Assume again that qualia inversion affects all colour perception. It follows from this assumption that qualia inversion will not be shown by any of the colour discriminations either of us makes. We will both call the grass green, the sky blue, and the stop sign red, even though all of those stimuli present different experiences to one person than to the other. In other words, your internal spectrum of colour experiences is *systematically* mapped onto the external world in a way that is precisely the *inverse* of my own. But, this internal difference is concealed by the fact that we use our shared colour *words* to external objects in all of the same ways.

The possible inversion of our respective colour qualia remains possible irrespective of how much we may know about each others' physiology, and irrespective of how similar we may be in our physical behaviour, our physiological make-up, and our physiological mechanisms. Indeed, our physiology could be *type identical* in every respect, yet our colour qualia could still differ; that is, qualia inversion would be physiologically undetectable. Since the physiological facts cannot reconstruct, as it were, the facts of experience, the mental facts must be some type of facts *above and beyond* the physiological facts. The argument then infers that the natural sciences cannot describe and explain mental experiences; so we must look to the realm of the *nonphysical* facts. Thus, inverted qualia reveal an unassailable *metaphysical mind-body gap* between the facts of mental experience and the facts of mechanism (Block, 1980; Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Jackson 1982; Ray et al. 2013; Searle, 1992).

A possible qualia inversion scenario can easily be introduced in an interpersonal context concerning pain: thus, you and I share the same range of experiences, are physically the same in all respects, but in all those instances where I have the experience of pain, you have the experience of pleasure. Once again, we could be exactly alike in physiology, yet our pain qualia could still differ. Since pain inversion would be physiologically undetectable, the facts about pain must be some type of facts other than the physiological facts (e.g., Block, 1980; Chalmers, 1996; Derbyshire & Raja, 2011; Searle, 1992).

I will offer two criticisms of the qualia inversion argument: the first criticism targets the truth of the key premise; the second criticism assesses the premise that conceivability entails possibility. If only one of these criticisms succeeds, then it follows that the qualia inversion argument does not succeed.

4.3.1 Is the Key Premise True?

What is the *key* premise of the qualia inversion argument? The key premise is the one that enables support for its conclusion, namely, 'Our brains could be identical in every respect, but our pain qualia could differ'. The first question I will ask about this argument is: is the key premise of the argument *true*? Given the compelling evidence that differences in personal experience do *in fact* involve differences in psychophysiological activity, the premise is not true with regard to pain. For example, pain at temperatures ~ 15°C occurs when 'HPC' (heat, pinch, cold) neuronal activity in C-fibres accelerates, while cooling-specific neuronal activity in Aδ fibres plateaus (Craig, 2003). Moreover, artificially blocking Aδ fibres enables nominally innocuous cool temperatures (up to 24°C) to produce intense *burning* pain (Craig et al. 1996). Direct repetitive transcranial magnetic stimulation (rTMS) of the precentral motor cortex using surgically implanted electrodes temporarily *relieves* medication-resistant neurogenic pain (Lefaucheur et al. 2001). The release of signal molecules such as opioid peptides and their receptors in the Arc-PAG-NRM-spinal dorsal horn pathway is pivotal in mediating acupuncture analgesia (Han, 2003). To my knowledge, there are no scientific reports of cases where the nervous system remains exactly the same but the personal experience of pain changes. Since there is a robust causal relationship between nervous system operations and the experience of pain, the *falsity* of the key premise of the qualia inversion argument is to be expected (Dennett, 2001).

Philosophers defending qualia inversion need to produce actual cases which yield the truth of the key premise. The argument requires a case where differences in pain qualia are physically undetectable; and not undetectable using only physiological detection techniques, but undetectable given physiological, anatomical, behavioural *and* psychological detection techniques. Moreover, these cases would need to be consililent with both established multilevel views of the physiological mechanisms that explain pain qualia, and situated

within the broader context of advancing physiological (e.g., nervous, endocrine, immune) theories *and* evolutionary (e.g., complex adaptive system) descriptions and explanations of pain experience. But, this strategy will be never be used by the supporter of qualia inversion because, as I have indicated, there can be simply *no evidence* to present on the matter; no genuine cases where nervous system operations are identical in every respect but pain qualia differ (Dennett, 2001).

Some philosophers have defended the truth of the key premise by claiming that qualia are *nonphysical experiences* (e.g., Derbyshire & Raja, 2011; Feinberg, 2012; Jackson 1982; Ray et al. 2013; Tye, 2006). Accordingly, it is thought, our physiology could be type identical in every way but our qualia could differ. However, powerful reasons for doubting this defense accumulate with the many detailed reports of the robust correlations between physiology and the personal experience of pain, or the related symptoms that commonly occur with it. Fever and sickness with pain, the *sickness response*, characterised by fever, malaise, fatigue, decreased appetite and libido, excessive sleep and difficulty concentrating is an immune system response (e.g., Elenkov et al. 2005; Wieseler-Frank et al. 2005). This response is cytokine-mediated and depends on the central nervous system (CNS). Following injury, macrophages and other cells release proinflammatory cytokines which act on the vagus and glossopharyngeal nerves, and the hypothalamus to cause a variety of unpleasant symptoms[3]. Wound-induced acute stress that fails to resolve and which leads to chronic pain is typically caused by dysfunctional recovery in the hypothalamo-pituitary-adrenocortical axis (HPA). The recovery process in the HPA involves the inverted U principle: too little cortisol (CORT) over time can cause persistent anabolism, too much CORT over time can cause persistent catabolism (e.g., Kloet, 2006)[4]. In both cases, loss of normal diurnal variation in CORT

[3] The sickness response is adaptive because it limits normal behaviour and social interactions and forces recuperation.

[4] Hypercortisolism is an indicator of severe depression (e.g., Wong et al. 2003). Chronic pain is briefly mentioned in Chapter Two, section 6.3.

pulsing has been identified with dysregulation (e.g., Kloet, 2006; Leistad et al. 2007, 2008). Thus, a dysfunctional endocrine recovery process is a physiological mechanism for chronic endocrine dysregulation. The rare pain phenotype congenital insensitivity to pain (CIP) is entirely dependent on the nonsense mutation of $Na_v1.7$, the α-subunit of the gene *SCN9A*, that is expressed mostly in nociceptive small-diameter dorsal root ganglia neurons (Cox et al. 2006; Cox et al. 2010). Chronic pain patients who have undergone a prefrontal lobotomy as a last resort for their intractable pain, show the typical reactions and symptoms when they are stimulated momentarily by normally painful stimuli, but they don't find such stimuli painful or aversive (e.g., Freeman et al. 1942; Hardy et al. 1952; Barber 1959). There are many more examples from the field of pain (e.g., Bonica, 1953; McMahon & Koltzenburg, 2005)[5].

Now, if the facts about pain constitute a realm of nonphysical facts, then why should cytokine and CNS mediated operations be type identified with the sickness response? Why should the mutation of $Na_v1.7$ *SCN9A* be type identified with CIP? Why should prefrontal lobotomy patients reliably report that normally noxious stimuli are not painful? The reason is that pain qualia, and qualia of pain-related symptoms, are *utterly reliant* on neurophysiology. This seems the simplest and most parsimonious description. Once again, philosophers defending qualia inversion need to present genuine cases which demonstrate the truth of the key premise consilient with established science. But it appears that there *cannot* be just such evidence.

The complete dependency of pain qualia on physiology also seems correct in light of the causal closure of the physical; namely, that every physical effect must already have an immediate sufficient *physical* cause is reason to think that anything nonphysical cannot make a causal difference to the physical (Papineau, 2002, 2009). At most, pain will be 'nonphysical' only in the trivial sense that it is characteristically referred to using specialist terminology in

[5] For a case not involving pain, see Churchland (2002) for a description of the disconnection effect (i.e., 'split brain').

the ‘nonphysical’ life sciences, and not because it is ontologically nonphysical. According to causal closure, apparently nonphysical pain experiences must themselves *in fact* be physical. Otherwise, how could they have any physical effects? The causal closure of the physical makes a strong case for *reducing* pain to the physiological: for it shows that anything that has a causal impact on the physiological realm must itself be physiological.

4.3.2 Does Conceivability Entail Possibility?

The qualia inversion argument attempts to challenge type identity theory by showing that physically undetectable inversion is possible. The simplest version of the qualia inversion argument, using pain, is this: (1) pain inversion is conceivable; (2) whatever is conceivable is possible; (3) therefore, pain inversion is possible. Now, imagine a pain inversion scenario enabled by the purely physiological facts, and physically detectable. This *anti-pain inversion* scenario seems conceivable. The simplest version of this scenario is: (1*) anti-pain inversion is possible; (2) whatever is conceivable is possible; (3*) therefore, anti-pain inversion is possible. However, (3) and (3*) cannot be both true: if the purely physical facts about *anti-pain inversion* make it physically detectable, then the same physical facts about *pain-inversion* make it physically detectable too, which means pain qualia cannot be ontologically nonphysical.

Consider a version of the analogous ‘zombie thought experiment’ (e.g., Chalmers, 1996): imagine a person *identical* to us in every way, but lacking pain qualia: such an individual would be a *zombie.* He would not feel pain. Since this scenario is conceivable, it is possible. Thus, zombies are possible. However, the same strategy used above in the case of pain inversion can be applied to the conceivability of zombies to generate an *anti-zombie* scenario: imagine identical copies of ourselves unable to experience pain by the purely physical facts. However, zombie and anti-zombies cannot both be true (e.g., Frankish, 2007).

Again, if the physical facts about *anti-zombies* cannot make them feel pain, then the same physical facts about *zombies* cannot make them feel pain too, which implies they are not zombies after all (Kirk, 1999). One consequence of this objection is that the inference from conceivability to possibility in the qualia inversion argument should be rejected.

On a related point, and to close this section, what *follows* from the fact that a philosopher who supports qualia inversion is, or is not, able to conceive how natural science could explain pain? Does anything informative concerning the nature of pain and its mechanisms follow from this fact? No; nothing especially informative. The inability to conceive how science could explain pain merely seems to add a fact about the *philosopher* – a fact about *him or her* – from which nothing informative follows about the nature of pain. More charitably, it is a fact about what is known or not known in contemporary science about pain. It is a fact about what we presently do and do not comprehend. It is a fact about what, using the extent of our comprehension, we can and cannot conceive. The mysteriousness of pain due to its seeming nonphysicality does not appear to be a property of *pain* itself (Churchland, 1996). To be fair, the belief held by some philosophers that metaphysical possibility commands to pain science and science generally what it can and cannot discover and explain – *ever*, appears doctrinal. Given these criticisms, the qualia inversion argument should be rejected.

4.4 Saul Kripke's Objection against Type Identity Theory of Mind

Saul Kripke's objection to type identity theory of mind should be viewed against the backdrop of his views on linguistic reference. Thus, I begin by briefly reviewing Kripke's philosophy of language. According to Kripke (1971, 1980), the reference of a name is initially determined by means of a baptism-like act, and is sustained over time through a kind of causal chain which ensures that the present application of the name is firmly connected to

the referent of the initial baptism. These causal features entail a metaphysical commitment, according to which the reference of a name is fixed by some *real essence* of the referent, which is a *necessary property*, without which the referent would not be what it is. However, in baptising and referring, we may not be presented with the real essence of a referent, but rather with some *nominal essence* of it; that is, with some *contingent property* of the referent. For example, the real essence of water is H_2O, but the nominal essence of water is the set of causal properties strongly correlated with it, such as being tasteless, thirst-quenching, colourless, and so on. Although the referent of 'water' is H_2O, we can infer water by inspecting for the presence of its nominal essence. However, the difference between real and nominal essences appears to vanish in the case of bodily experiences such as pain. For, according to Kripke, there is nothing in pain which is *not* in apparently feeling pain. If it appears to me that I am in pain, then there is nothing else that I must instantiate in order for me to be in pain. Thus, in the case of pain and other bodily experiences, nominal and real essences *coincide*: all the properties that make pain the experience it is, are necessary to it.

Kripke's (1980) views on linguistic reference are continuous with a metaphysical scenario he makes in *Naming and Necessity* (1971) against type identity theory. According to this scenario, pain cannot be type identical to any physical state because having pain in an individual without any specific type of neurophysiological state being invariably tokened is *conceivable*. It is also *conceivable* for *two* mental state type identical individuals to be different as to their neurophysiological states. Since this scenario is conceivable, and conceivability implies possibility, it follows that mental states cannot be type identical to neurophysiological states. Therefore, type identity theory of mind is false. To fully understand and assess this sketch of Kripke's objection, I will consider an argument I call *Kripke's Qualia Argument*.

4.4.1 Kripke's Qualia Argument

According to Kripke, qualia type mental states such as pain *necessarily*: 'Pain is not picked out by one of its accidental properties; rather it is picked out by the property of being pain itself, by its immediate phenomenological quality' (1980: 152). Since Kripke thinks that nominal and real essences coincide in some mental states, if pain is necessarily picked out by its immediate phenomenological quality; the name 'pain' refers to this quale and not to any strongly correlated casual property such as facial grimacing. When it comes to pain, what you feel is what you have. Appearance and reality coincide. In addition, the word 'pain' refers to this same quale in every possible world; that is, 'pain' is a *rigid designator* (Kripke, 1980).

In responding to Kripke's argument, the type identity theorist can accept Kripke's premise that *appearance* refers to the quale that is immediately present to the mind in a pain episode, and add the claim that the *reality* of pain refers to the specific physiological operations of the NEIM (Chapman et al. 2008; Chapman, 2010; Melzack, 1999). Thus, I will assume that appearance and reality in an episode of pain coincide. However, this does not rule out the metaphysical possibility that pain qualia are type identical to specific operations of the NEIM (e.g., Hill, 1991). In the case of pain, appearance and reality are *type identical*. If the name 'pain' refers to a pain quale, it also refers to the operations of the NEIM, which is type identical with the presented quale. Thus, neurophysiological properties are necessary properties of pain.

Kripke (1980) replies to this objection as follows. If pain were type identical to the operations of the NEIM, and 'pain' and 'the operations of the NEIM' are rigid designators, then their identity would be *necessary*, since identities – if true – are necessarily true, even if not *a priori* (Kripke, 1980). We might initially think that since the identity claim 'pain = NEIM operations' was only found empirically to be the case (Chapman et al. 2008; Chapman, 2010; Melzack, 1999), it must be *contingently* true. But, says Kripke, it is necessarily true.

To clarify Kripke's thinking, consider the type identity statement 'Hesperus = Phosphorus' (Kripke, 1980). This type identity description was found *a posteriori* to be true. 'Hesperus' is a name that was assigned to a heavenly body viewed in the evening, and 'Phosphorus' is a name that was assigned to that same heavenly body viewed in the morning. The heavenly body is Venus. According to Kripke (1980), the identity description 'Hesperus = Phosphorus' is necessarily true because 'Hesperus' and 'Phosphorus' are proper names for the same referent. Both names are rigid designators: each designates just the referent it *actually* designates in all possible worlds in which that referent exists, and it designates nothing else in any possible world. The referent in question is Venus. Since 'Hesperus' and 'Phosphorus' both name Venus in all possible worlds, and since Venus = Venus in all possible worlds, 'Hesperus = Phosphorus' is true in all possible worlds.

By contrast, a description like 'the brightest non-lunar object in the evening sky' is not rigid (Kripke, 1980). The identity statement 'Hesperus = the brightest non-lunar object in the evening sky' is true, but not *necessarily* true, for although it designates Hesperus in this world, which explains why it is true, the description might have designated Mars in some other worlds, which explains why it could have been false. That is, the identity 'Hesperus = the brightest non-lunar object in the evening sky' would have been false had some other such world been actual. While Hesperus is in fact the brightest object in the evening sky apart from the moon, it is conceivable that Mars might have been the object designated by 'the brightest non-lunar object in the evening sky' rather than Hesperus. Hence, the identity claim could have been false because 'the brightest non-lunar object in the evening sky' does not designate Hesperus *rigidly*.

I now return to the initial objection given above, and Kripke's response to it. Assuming pain is *necessarily* type identical to the operations of the NEIM, Kripke thinks that we can *conceive* a scenario in which an individual is in pain and yet does not have his NEIM

active. Moreover, we can also conceive a case in which a person has her NEIM operating and is not in pain, because the operations of that neurophysiological mechanism should not necessarily be felt as pain. Since this scenario is conceivable, and conceivability implies possibility, it follows that pain qualia *cannot* be type identical to neurophysiological states.

To illustrate the point, Kripke considers identifying heat with an experiential quale. Can we conceive an individual presented with the same quale we have when we feel heat and yet she does not feel heat? The answer is affirmative, Kripke maintains, because *feeling* heat is not a necessary property of heat. It is conceivable that there are *possible worlds* in which heat exists but in which there are no conscious beings. When we conceive an individual who has the same quale we have when we feel heat, we are in fact conceiving an *epistemic* episode which is qualitatively type identical to the one in which we feel heat. But what the imaginary individual is feeling is not heat *per se* so much as the quale reliably correlated with heat. According to Kripke, the scenario with heat doesn't hold in the case of pain, for while the quale that we have when we feel heat is not a necessary type property of heat, the quale we have when we are in pain *is* pain. Thus, an epistemic episode qualitatively type identical to one in which we are in pain is one of pain. Whereas pain is designated by its immediate quale, the operations of the NEIM are designated by the neurophysiological and causal structure of the brain and body.

4.4.2 An Objection to Kripke's Qualia Argument

I will now advance an objection against Kripke's argument. Essentially, I claim that Kripke's argument against type identity theory of mind assumes that qualia are epistemically *subjective* yet ontologically *objective*. I will argue that ontological objectivity is consistent with the type identity theory of the mind.

In the case of pain, assume that appearance and reality coincide. A reason for making this assumption into a phrase of art is that the type identity theory of mind is committed to honouring philosophical intuitions about qualia, despite being empirically insufficient[6]. Since nothing else can determine the reality of a pain quale, the type identity of a pain is determined by the conscious experience of the individual who feels it. If the appearance of a quale necessarily relies on conscious experience of a quale, then qualia are *essentially subjective*; that is, they rely entirely on the consciously feeling individual. However, a given experience of a pain quale does not provide us with the means to determine whether a pain quale is of the *same type* as another one that occurs at a *different time*. Any such means would have to specify that pain qualia are fundamentally independent of the way they are subjectively experienced to be at different times. However, this premise entails that the means would have to specify that pain qualia are *objective* and thus *independent* of their presentation to individuals.

Similarly, if *two different individuals* have the same type of mental state, then these states, since they are type identical irrespective of who has them, are *no longer* essentially subjective, even if they are only epistemically accessible through the conscious experiences of the individuals concerned. Since we cannot directly access pain in other individuals, and such states are not essentially subjective, then the only way we can conceive different individuals who are both in pain is to consult some objective criterion for when they are *feeling that way*. The best description for this objective condition is that the two individuals are in the identical type of *physiological* state. That is, the physiological state being the operation of the NEIM. Thus, if mental states fall within a type, there must be an objective type identity criterion by which we can describe and explain whether the same individual, or

[6] In fact, it is false. Patients with *pain asymbolia* don't respond to even acute pains like pinpricks, minor cuts, or burns. Experimental pain stimuli fail to generate any identifiable aversion. However, the patients insist that the stimuli cause pain: they identify their experiences as pain (e.g., Berthier et al. 2004; Grahek, 2007, Rubins & Friedman, 1948). I will discuss pain asymbolia in Chapter Five in relation to eliminative materialism.

two individuals at different times, have the same type of quale. If this reasoning is correct, a supporter of Kripke's anti-identity argument faces two options: either reject the coincidence of appearance and reality in the case of pain and qualia in general, *or* accept that pain is type identical with the operations of the NEIM.

To clarify this objection to Kripke, I will say more about the nature of type identity. Assume that the experience of pain is what it is because of the presented quale: there is nothing in a pain quale which is independent of how it feels to the individual of the experience. However, since appearance and reality coincide in the experience of pain, it follows that there is no difference between a pain quale as it is experienced and the experience of a pain quale. For example, it is not possible for me to have *this* experience of pain – to have another experience of the very same pain quale – because that would be a different experience. In accepting that the appearance and the reality of pain are identical, a supporter of Kripke's anti-type identity argument also accepts that qualia and the experience of qualia are type identical, since the only way to have qualia is to directly experience them. Thus, the experience of a pain quale appears to be private and incorrigible in the strong sense that no one else can epistemically access my pain in the way I have direct access to it; namely, by feeling it and coming to know I am feeling it on that basis.

Now, in order for two individuals to have the same type of pain quale, it is necessary for them to have the same type of experience. Experience and the tokening of qualia cannot be separated; so the two individuals will have one and the same type of pain quale. Thus, for two individuals to token the same type of pain quale, they must have the same type of experience of a pain quale of the same type. However, this requirement appears to imply that the experience of pain cannot be essentially subjective: if it is given that you and I can have the *same* type of pain quale, and therefore have the *same* experience, it follows that a quale cannot be essentially subjective, and cannot explain the experience of a pain quale. The

reason is this: if pain qualia can be invariably felt by several (conscious) individuals, then its identity is *independent* of those individuals, even though qualia rely on conscious individuals in order to be experienced. Similarly, if you and I have the same type of pain quale, our experiences cannot determine the quale tokened, since once again, there is no difference between the experience of a quale and the quale experienced. Thus, we token the same type of experience, which implies that the experience of pain is *not* essentially subjective. The concepts of qualia and conscious experience can clarify how two individuals can have the same type of quale. This means I reject Kripke's assumption that pain is how it feels to an individual to be in pain.

If my argument is correct, it follows that if an individual or individuals have pain at different times, then these states cannot be essentially subjective, for it is conceivable for two or more individuals to have the same quale (appearance) in virtue of having the same experience. Having the *same* pain cannot be a *pain*. But if pains are essentially subjective and private, then every individual may personally establish different type identities for pain qualia, derailing the possibility of a type identity theory of pain. Thus, it appears that an objective criterion is required to establish these type identities. If two individuals have the same type of quale, then the quale in question is objective. Again, since one individual cannot directly access another individual's pain, there must be an objective type identity criterion by virtue of which it can be judged that two individuals are in fact having a type-identical pain. The possibility of their type identity relies on a criterion which is *independent* of epistemic access to pains. Thus, the nature of pain qualia should be established objectively. The only way to determine the referent of the term 'pain' is to assume that in baptising this type of state we are also referring to the type of neurophysiological mechanism by virtue of which the quale remains unchanged in its various presentations; that is, the NEIM.

The Kripkean philosopher is open to respond to my objection. A Kripkean may say that an objective type identity criterion would only be required in order to *verify* whether two individuals are having the same experience, and not if we merely want to *conceive* it. Suppose the type identity theorist agrees with the Kripkean philosopher to deny that the objective type identity criterion is required to conceive two individuals having the same pain quale. This concession, however, appears to create a contradiction. Consider that you and I are both in pain and can, simultaneously, directly access each other's pain. Assume further we both *feel* we have the same type of quale. According to the coincidence of the appearance of pain and the reality of pain, we both have the same mental state. But now consider this scenario: I feel we have the *same* type of pain, while you feel we have *different* types of pain; that is, you feel the two states are different. Are we in the same type of mental state or not? Since there is nothing to the reality of pain other than the way it is felt, then we both have *and* do not have the same type of pain. Since it is conceivable that we diverge (feel differently) concerning our pains, if we reject an objective criterion of qualia type identity, then we generate a logical contradiction.

It is time to sum up my objection to Kripke's argument. It appears that tokening the same type of pain quale at different times in one or two individuals requires an objective criterion for its type identity. The same pain quale cannot be both the tokening and the criterion; however, since it would in that case be a *personal subjective criterion*, whose correct application only the individual or individuals could attest to. Similarly, conceiving two individuals in pain independently of their feelings requires an objective criterion of type identity. If this type identity is based on the feelings of both individuals, then the contradiction described in the previous paragraph follows. Kripke's argument against type identity theory of mind assumes that qualia are epistemically *subjective* yet ontologically

objective. I have argued that ontological objectivity is consistent with the type identity theory of the mind.

5 Conclusions

In this chapter, I have attempted to make the case for type identity theory of pain by assessing type identity theory of mind. I offered five reasons to prefer type identity theory to alternative philosophies of mind: it has greater explanatory power; it is more respectful of philosophical intuitions about the causal powers of qualia; it is simpler, it is supported by causal closure of the physical; and it is continuous with natural science. I described and challenged four 'conceivability' objections to type identity theory of mind: mental states are excluded; mental states disappear; inverted qualia; and Kripke's assertion that type identity theory is false because two individuals could have the same mental state while having different physiological states. We should also note that the cogency of conceivability arguments is an important question in contemporary philosophy. While conceivability arguments do present challenges to type identity theory of mind, in fact to all identity theories of mind, they are mostly directed at physicalism. Thus, it is unclear whether type identity theory must make special provision to address objections based on conceivability arguments.

It might be complained that the analysis I have provided in this chapter is overly general, and a detailed description of a type identity theory of *pain* is required in order to describe and explain the nature of pain, this being the fundamental ambition of the project. So, in Chapter Two, I offer a well established descriptive metaphysic for a type identity theory of pain.

2 Bridging the Metaphysical Mind-Body Gap: A Type Identity Theory of Pain

Abstract

What is pain? The aim of this chapter is to offer the best description of this *descriptive* mind-body question in terms of a type identity theory of pain backed up by a general and robust theoretical schema. Pain is a multidimensional experience and involves specific sensory, emotional and cognitive features. I frame a well established multilevel description of the physiological mechanisms that best describe pain qualia within the context of advancing descriptions of the nervous, endocrine and immune systems and their functional interdependencies, and descriptions of allostasis, homeostasis, stress and wounds, all constrained in turn by complex adaptive systems theory. A biological individual is a complex adaptive system coping with a physical and social environment, but possessing nested subsystems. These descriptions in concert best reveal how pain qualia are type identified with mechanism. Somatosensory qualia of pain, including submodality, intensity, duration and location, are the operations of multisubsystem mechanisms (neospinothalamic tract); negative emotional pain qualia are the operations of multisubsystem mechanisms (paleospinothalamic tract); cognitive pain qualia (pain anticipation) are the specific operations of the primary somatosensory cortex (neospinothalamic tract); and pain suppression (stress-induced analgesia) is the operation of the dorsolateral funiculus pathway and opiate systems (paleospinothalamic tract). The simplest, most coherent and parsimonious explanation of these relationships is that pain is an allostatic stress mechanism comprised of interdependent nervous, endocrine and immune operations. Pain is mechanism.

Keywords: allostasis, biological individual, chronic pain, complex adaptive system, dysregulation, homeostasis, mechanism, mechanistic description, nervous-endocrine-immune mechanism (NEIM), pain, qualia, stress, type identity theory of mind, wounds

1 Introduction

The aim of this chapter is answer the fundamental descriptive puzzle of the mind-body problem: *what* is pain? I claim that the state *being a pain* is identical with the state *being the operation of specific physiological mechanisms*. What establishes this metaphysical thesis? I propose that the answer to this question is that if *x* is identical to *y* then *x* and *y* have the same type identity *conditions* (Sidelle, 2008; Wetzel, 2008). Type identity conditions are

> *the sorts of things that are represented by statements saying, for any possible object, what features it must have in order to be, or those which suffice for it to be (identical to) some particular thing (or, for kinds or properties, for something to be a member of that kind, or possess that property)…a specification of identity conditions need not state with full precision – need not mention – what the relevant features are; for certain purposes, 'this chemical microstructure' or 'this thing's origin' will do as well as 'H2O' or 'sperm S and egg O'.* (Sidelle, 2008, p. 291)

In other words, if two types of things have exactly the same type identity conditions, then they are the same type of thing. Thus, determining that two things have the same type identity conditions is sufficient for determining their type identity (Sidelle, 2008; Wetzel, 2008). Applied to the project thesis, pain and physiological operations are type identical if they have exactly the same type identity conditions. The type identity conditions of pain and physiological operations can be given when we have detailed delineations both of pain *experience* (i.e., pain qualia), and of physiological *mechanisms*. Once such delineations are available, the best description of them is that the described experiences and neurophysiological mechanisms have exactly the same identity conditions; that they are the same thing (Sidelle, 2008; Wetzel, 2008). The more we can delineate pain experience and

mechanism, the greater the likelihood for metaphysical description. The availability of explanation in turn means that what might escape description gradually shrinks towards zero (Sidelle, 2008; Wetzel, 2008).

Traditional type identity theories claim that sensations are brain operations, but they do not make any metaphysical assertions concerning the nature of brain operations (Polger, 2011). In this project, I will break with this tradition. I propose that a metaphysical description of how pain experience and its physiological operations are type identical involves *mechanistic descriptions*. I now briefly review the core concepts of mechanism and mechanistic description, as they are understood in contemporary philosophy of science. I also describe the required task of situating such descriptions in a general metaphysic that can best unify scientific descriptions. The overarching framework theory I have selected is *complex adaptive systems theory*.

2 Mechanism and Mechanistic Description

In recent years, some philosophers of science have turned to understanding mechanism delineation as the approach to scientific description in biology (Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993; Craver, 2007; Darden, 2006; Glennan, 1996, 2002; Machamer et al. 2000; Sarkar, 1998; Schaffner, 1993; Woodward, 2003). There are differences between the various descriptive philosophies of a mechanism. Some philosophers have focused on the decomposition of a complex system into interacting component parts and operations (e.g., Bechtel & Abrahamsen, 2005; Bechtel & Richardson 1993; Glennan, 2002); other philosophers have maintained the importance of inclusion under general rules or *laws of nature* (e.g., Sarkar, 1998; Schaffner, 1993). But they all share the fundamental descriptive claim that a scientist offers a successful description by identifying and manipulating variables in a uniform causal mechanism thereby establishing how those variables are situated in and

make a difference in the mechanism. The ultimate description amounts to the delineation of how those variables act and interact in order to generate the phenomenon of interest (e.g., pain). A delineated mechanism enables the descriptive and explanatory features of understanding, control, and prediction. I favour the philosophy of scientific description and explanation in biology proposed by Bechtel and Abrahamsen (2005) and Bechtel and Richardson (1993) because it is truest to the approach of pain researchers when engaging in physical description and explanation. Pain researchers describe their practice and achievements as the delineation of physical mechanisms rather than laws of nature. I elaborate on this specific issue in sections 4.1-4.4 of Chapter Three.

A core feature of all contemporary philosophies of mechanistic description is the assertion that a mechanism consists of component parts that perform operations which are organized in order to produce a phenomenon (e.g., the experience of pain). Identifying parts and operations consists in *decomposing* a mechanism either structurally into its component parts or functionally into its component operations (Bechtel, 2002; Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993). In scientific practice, the task of decomposition involves either taking the actual mechanism apart, or taking it apart in theoretical analysis. An important aspect of decomposition is *localizing* component operations in the component parts that perform them (Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993). Localization claims are identity claims between the component parts delineated according to their physical make-up (e.g., nociceptors delineated according to thin myelination or unmyelination) and component parts delineated according to the component operations they are involved in (e.g., initiating neurogenic inflammatory processes that amplify responses to noxious or innocuous stimuli). These type identity claims are one level *down* from the initial identity claim correlating a phenomenon (e.g., pain) to the mechanism generating it. Mechanistic descriptions are *ontologically reductive* because decomposing a mechanism into its

component parts and component operations consists in going to a *lower* level of actual organization (Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993). These features of mechanistic description are discussed in Chapter Three with regard to scientific explanation.

For the delineation of the parts and operations of a mechanism to best describe a phenomenon, we must be able to follow how those parts and operations work together in concert to generate that phenomenon. A core feature of this is to learn the component operations, which are *processes of change* (Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993)[7]. A further aspect is to understand how the parts are organized and their operations coordinated. The functioning mechanism is thought to involve sequential performance of component operations 'from start or set-up to finish or termination conditions' (Machamer et al. 2000). However, many biological mechanisms involve *non-sequential organization,* where the individual operations are *non-linear* when delineated mathematically and operate in an environment that is open to flows of energy. Under these conditions, mechanisms can manifest *dynamic behaviour*. To understand this dynamic behaviour, Bechtel and Abrahamsen (2010) propose integrating tools of mathematical modelling and mechanistic research, generating what they term *dynamic mechanistic descriptions*. A key aspect of dynamic mechanistic descriptions is the use of mathematical models (e.g., multivariate statistical analysis) to understand what operations (e.g., positive and negative feedback loops) are produced by the mechanism.

A canonical example of a physical mechanism is DNA replication. As Watson and Crick (1953) observed following discovery of the structure of DNA, the macromolecule's structure suggested the mechanism of DNA replication: 'It has not escaped our notice that the specific pairing we have postulated immediately suggests a possible copying mechanism for the genetic material'. In other words, the double helix of DNA (a component part) unwinds (a

[7]. Some philosophers delineate the way in which the parts of a mechanism behave as an *activity* (Machamer et al. 2000), an *interaction* (Glennan, 2002; Woodward, 2003), and an *interactivity* (Tabery, 2004).

component operation) and new component parts bond (a component operation) to both parts of the unwound DNA helix. DNA is a nucleic acid comprised of multiple subparts: nucleic acid bases and a sugar-phosphate backbone. When DNA unwinds, the bases manifest weak charges. The weak charges result from small molecular asymmetries, and operate to enable a DNA base and its complement to form hydrogen (weak polar) chemical bonds. The specificity of this operation is best described as the geometric organization of the weak polar charges in the subparts of the base. Ultimately, component parts with polar charges allow the operation of hydrogen bond formation. After the complementary bases align, then the backbone forms via stronger covalent bonding. The mechanism continues with unwinding and bonding together (operations) new parts, to yield two helices (new parts) that are approximate copies of the parent helix.

2.1 General Framework Theory: Complex Adaptive Systems Theory

In addition to localizing operations in the parts that perform them, mechanistic description must also be localized in the broader framework of general theories that unify scientific descriptions and explanations (Churchland, 1989; 2002). Once a mechanistic description is situated in a general descriptive theory, type identifications are implied (Churchland, 1989, 2002). To illustrate this idea, consider the physical theory of temperature (Lyons, 1985). By the middle 1700's, the experiments of Daniel Fahrenheit (1686-1736), Joseph Black (1728-1799) and others had established a quantitative calorimetric of temperature as heat flow and capacity; but this didn't really describe just *what* was flowing. Thus, formulating a precise description of 'heat' at the time was not possible, since physicists did not know what it really was. Certainly, no physicists could say that heat created through friction was micromechanical motion; since it was considered observably obvious that it was a type of *invisible fluid* that moved from hot bodies into cold bodies. Such precision as there

was in defining ‘heat’ consisted in grouping together a range of well-known experimental effects and observations, and inferring the physical properties of temperature change: since heat flowed from hot bodies into cold bodies and spread throughout the body, obviously its particles repelled each other, just like those in electrical fluid; however, in contrast to electricity, which had no observable effect on the appearance of a charged object, when heat was added to a solid object, it changed considerably: first, the material expanded, then it changed to a liquid and finally to a gas, if sufficient heat could be provided. Further heating expanded the gas, or increased its pressure if it was held in a fixed container. To interpret this sequence of events in terms of a caloric fluid being inserted into the material, the physicists imagined the fluid flowing between the atoms of the solid and lessening their attraction for each other, until the solid melted into a liquid, whereupon the caloric continued to accumulate around the atoms until they were pushed apart into a gas. John Dalton (1766-1844) proposed that in the gas each atom or molecule was surrounded by a ball of caloric, like a springy ball of wool, and these balls were packed in a container like oranges in a crate, except that the caloric balls could expand *indefinitely* as heat was poured in. Various other effects were described by caloric theory: a suddenly compressed gas gets hotter because the same amount of caloric is now occupying a smaller volume; rubbing two solids together jostled the caloric fluid that was normally sequestered in the spaces between atoms, the jostling allowed the caloric to escape, or perhaps tiny pieces of material are rubbed off, and lose their caloric; thus heat appears. The criteria were drawn from what seemed intuitively obvious: they all involved movement of a conserved quantity, as would be expected of a fluid.

Heat created through friction was a problem, because there was no evident fluid *source* of caloric. To test the solution to the friction problem, Count Rumford Benjamin Thompson (1753-1814) travelled from England to a factory in Bavaria that bored holes in iron cannons. The cannons were bored by turning an iron bit inside a brass cylinder. The boring

continuously produced a huge amount of heat through friction of the iron bit on the brass. It was conventionally thought that the pressure and movement of the boring released caloric fluid in the fragments that were sheared off. Rumford hypothesized that if caloric fluid was squeezed out by friction during boring, the caloric fluid should eventually dissipate. No extra heat should be produced by further boring or rubbing. Rumford observed that heat never ceased to be produced as the holes down the cannon shaft were continuously bored. At no time did the caloric fluid in the iron show any sign of dissipation. Rumford reasoned that either there was an *indefinite* amount of caloric in the iron, or the theory itself was fundamentally flawed. He prudently judged the first option to be empirically impossible. Assuming it *is* possible, even one's hands would have to contain an indefinite amount of caloric, since a person can keep rubbing them without dissipation in heat production. Rumford inferred that not only was caloric fluid not a basic type of fluid; it was not a fluid of any type. Heat relied on a completely different fundamental description. Heat, he described, just *is* micromechanical motion.

Caloric theory was eventually rejected because its contact with other parts of established science became worse rather than better, and because it proved inferior in descriptive and predictive power to the theory that heat is molecular motion, which meshed better with known science. These elaborations resulted in the differentiation between heat (energy transfer as a result of difference in temperature) and temperature (movement of molecules). Could we accept the physical theory of temperature and still doubt whether temperature is molecular kinetic energy? No. Once we accept the theory of kinetic energy, the type identity of temperature naturally emerges as a result. It appears this way because the theory of kinetic energy determines the *type identity conditions* for the motion of particles (atoms, subatomic particles, molecules). Further, we cannot ask whether temperature could have different properties that it does, since the theory of kinetic energy best describes the properties of energy in terms of their type identity constituting particle structure. Once we

accept kinetic theory, the descriptively powerful framework thereby assumed makes no provision for delineations such as these[8].

The case of bodily experiences and physical operations appears no different than the case of kinetic energy and temperature. In actual practice, however, the case is not straight-forward, because neurophysiology is still in search of its fundamental descriptive and explanatory exoskeletons; of the general descriptions and principles that describe and explain how nervous, endocrine and immune systems function and in which to frame mechanistic descriptions of bodily experience (Churchland, 2002, 2011). Although there are many accurate descriptions of the structure and the function of individual neurons, hormones, and lymphocytes, the basics delineating how macro effects materialise from these cells and chemicals are still largely undescribed. While it is difficult to predict the course of a science, and especially difficult to predict what a young science will look like when it matures, the absence of fundamental neurophysiologic descriptions and explanatory principles is not positive evidence for something else, such as nonphysical qualia. As it turns out, there *is* a very broad descriptive theory for biological and psychological sciences that can offer the best *general* description, and that is *complex adaptive systems (CAS) theory*. Using CAS to constrain our descriptions of pain can help to establish the mechanistic descriptions and explanations in the theory. Since the interactions of nervous, endocrine, and immune operations to wounding are complex and adaptive, a systems approach is well-positioned to advance understanding of pain.

In itself, CAS theory does not entail brain-mind identities, but it can provide a *general descriptive framework* for theories that have this purpose. In this way, what I am offering is something like what Schaffner (2006) terms a 'patchy and fragmentary' mechanistic metaphysic description, rather than a final or completed mechanistic description.

[8] This does not imply that the nonexistence of caloric fluid has been absolutely proven, but because this concept plays no descriptive and explanatory role whatever in science, it should be regarded as an outdated theoretical description.

Neurophysiologists provide mechanistic descriptions even if many facts have not yet been described and present explanations still require more details to be filled in (Baars, 2012; Churchland, 1989, 2002; Demertzi & Laureys, 2012). Current neurophysiology effectively describes individual facts and generalities of smaller scope, and the *explanans* can be a mere sketch of a theory (Baars, 2012; Churchland, 1989, 2002; Demertzi & Laureys, 2012). To that extent, fragmentary mechanistic descriptions are sufficient for the task at hand.

I intend this descriptive metaphysic to be quite general. To illustrate, take the conventional example of the type identification of fire and rapid oxidation. Why is this type identification descriptive (i.e., informative)? The first step is to conduct a qualitative investigation of fire. The flame is the visible part of fire, it releases heat and light, is normally sustained by a continuous supply of fuel, and so on. Some qualitative facts about fire are easily observed and others take further investigation, for instance, facts about the reactions that make fire explode. This provides a provisional description of fire. These qualitative descriptions (facts) about fire are then matched with qualitative descriptions (facts) about the operation of rapid oxidation, which is the sequence of chemical reactions between a fuel and an oxidant, such as oxygen or fluorine gas. These facts are harder to describe but essential. When *sufficient information* is at hand concerning the parts and operations of fire and the parts and operations of specific chemical reactions (rapid oxidation), we can describe how the structure of fire delineates its qualitative chemical properties. The *multilevel* mechanistic description of fire type identifies it with a specific mechanism type, rapid oxidation, and describes its behaviour in terms of the behaviour and composition of this mechanical operation. Fire is rapid oxidation. The fire tetrahedron is a systematized diagrammatic description of the fundamental type identities and properties best described by the multilevel mechanistic description of fire.

The type identification of fire and rapid oxidation is only enabled if other substances

are also type identified with other molecules, and if elements are type identified with chemical types, and so on. That is, the type identity of fire and rapid oxidation works because it is framed in the broader *descriptive context* of chemistry and physics. Those general framework theories imply the type identifications. Of course, the type identification of fire and rapid oxidation might be faulted as an incorrect description, perhaps because the physical operations involve activity in a broader range of physical processes. But that criticism merely asserts a different type identity description, and does not challenge type identity claims *per se*. This is the point I made previously concerning temperature and molecular kinetic energy. It *is* conceivable to ponder whether fire is *correctly type identified* with rapid oxidation rather than with some other operation; but within the framework of chemistry and physics as they are understood, it is not reasonable to ponder whether fire might fail to be any type of mechanical operation *at all* (Baars, 2012; Churchland, 1989, 2002).

In the remainder of this chapter, I apply this descriptive metaphysic to pain. The personal experience of pain is multidimensional and involves specific sensory, affective and cognitive features. There is a well established multilevel view of the physiological mechanisms that best describes pain qualia. This mechanistic description is framed within the context of advancing theories of the nervous, endocrine and immune systems and their complex functional interdependencies. There are also complex adaptive system-based descriptions of pain experience. Taken together, these descriptions reveal how pain qualia are type identified with mechanism.

3 Pain in the Brain?

The official scientific definition of *pain* was initially formulated in the 1980s by a committee organized by the International Association for the Study of Pain (IASP). This

definition was updated in the 1990s by the IASP to reflect advancements in pain science and has since been widely accepted by the scientific community:

Pain: An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note: The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment. Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain but are not unpleasant, e.g., pricking, should not be called pain. Unpleasant abnormal experiences (dysesthesias) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain. Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain, and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause (IASP-Task-Force-On-Taxonomy, 1994: 207-213).

An apparent immediate and inconvenient fact facing type identity theory of pain is that pain stubbornly resists identification with only the brain. The original pain identity statement, 'Pain = C-fibre activation' (Place, 1956), neglects two essential features of pain observed in contemporary pain science: (1) Conscious awareness of wounding is *multimodal* and is correlated with integrated visual, kinaesthetic, and enteric sensory modalities in addition to noxious signalling (e.g., Chapman et al. 2008); (2) Wounding is typically part of overall bodily awareness that is correlated with *multiple* reciprocal nervous, endocrine and immune states (e.g., Chapman et al. 2008; Lyon et al. 2011; Vierck et al. 2010). Convergent lines of evidence demonstrate that wounding followed by pain is strongly correlated with endocrine and immune operations as well as sensory signaling that together exert an extensive *non-neural* impact. These operations interact and comprise a defensive stress response to wounding[9]. I will articulate the fundamental background theories of these core concepts in section 6 below.

A consideration of the *higher* structures of the central nervous system (CNS) alone reveals an extraordinarily *complex* picture of pain. Unimodal functional brain imaging studies of nociceptive transmission, projection and processing show that signals of wounding reach higher CNS levels via the spinothalamic, spinohypothalamic, spinoreticular pathways (i.e., the paleospinothalamic tract) including the locus caeruleus (LC) and the solitary nucleus, spinopontoamygdaloid pathways, the periaqueductal gray (PAG), and the cerebellum (e.g., Burstein et al. 1991; Price, 2000). The thalamus (THA) projects to limbic areas including the insula and anterior cingulate, which have been identified with the integration of the emotional and motivational features of pain (Craig, 2002, 2003a, 2003b). Noradrenergic pathways from the LC project to these and other limbic structures. Accordingly, pain reveals *extensive* limbic,

[9] In clinical settings, problems of acute and chronic pain do not easily conform to pain-brain type identities. The persistence of chronic pain as a major problem in medicine may indicate that identifying pain with the brain ('pain in the brain') has failed to inform clinicians toward curative interventions (e.g., Chapman et al. 2008).

prefrontal and somatosensory cortical components. A meta-analysis of the literature described brain operations during pain as a complex network involving THA, primary and secondary somatosensory cortices (S1, S2), insula (INS), anterior cingulate (ACC), and prefrontal cortices (Apkarian et al. 2005). Thus, the brain engages in massive, distributed, parallel processing in response to noxious signaling.

The mechanisms of *multimodal integration* pose a formidable challenge for pain scientists. Hollis et al. (2004) examined how catecholaminergic neurons in the solitary nucleus integrate visceral *and* somatosensory information when peripheral inflammation is present. Pre-existing fatigue, nausea, intense physiological arousal, and a systemic inflammatory response induced by proinflammatory cytokines (e.g., Anderson, 2005; Eskandari et al. 2003) are all correlated with sensory signalling in the experience of pain. In addition to Craig (2002, 2003a, 2003b), an increasing number of studies have investigated the integration of information from multiple sensory modalities and central operations correlated with emotion and cognition in pain (e.g., Bie et al. 2011; Liu et al. 2011; Neugebauer et al. 2009). The more we are able to delineate the qualia of pain and map these experiences onto specific multimodal physical operations, the closer we come to identifying pain with those operations.

So, why has Place's (1956) original pain identity statement survived in philosophy of mind? One reason is that the use of 'C-fibre activation' by identity philosophers is merely a *placeholder* for whatever the eventual mechanisms of nervous systems prove to be. We now know that wounding is identical to specific endocrine and immune operations *in addition to* sensory signaling. These operations interact and in concert comprise a defensive stress response to wounding. However, the *purpose* of calling it the identity theory of *mind* is to separate it from philosophical theories that identify mental states with states of immaterial souls or minds (dualism), abstract machine systems (functionalism), or those theories that

reject the reality of mental states (eliminativism). It is not to make any substantive assumption about the sensory modality. This is why Place's (1956) pain identity claim of C-fibre activation has survived, despite being explanatorily incomplete.

4 Overview of a Type Identity Theory of Pain

Pains are complex experiences mechanically decomposed according to somatosensory, negative emotional and cognitive qualia (e.g., Chapman et al. 2008; Hadjistavropoulos et al. 2011; Hill, 1991; Melzack, 1999). I claim that pain qualia are best described as type identical with the operations of specific physiological mechanisms. *What* is pain? Pain is mechanism. The somatosensory features of pain include submodality, intensity, duration, and location (e.g., Melzack & Casey, 1968; Price & Dubner, 1977a, 1977b). The negative emotional aspects of pain include, but are not limited to, unpleasantness, fear, anxiety, anger, and shame (e.g., Lumley et al. 2011; Hadjistavropoulos et al. 2011; Melzack, 1999). Cognitive pain phenomena include beliefs, attention, memory, decisions, appraisal, thoughts, and anticipation (e.g., Eccleston & Crombez, 1999; Hadjistavropoulos et al. 2011; Price, 1999).

I do not deny that pain has features that initially do not *appear* to be type identical to mechanism; features such as belief, anticipation, shame, and also complex related social phenomenon such as pain empathy (van Rysewyk, 2009) and synesthesia (Fitzgibbon et al. 2010), at least in part because evidence to substantiate their fundamental type identities is lacking. However, *sufficient evidence* already exists to show a type identity of pain better describes pain, compared to dualism; even though the evidence has not yet type identified all features of this descriptive metaphysic. Thus, I believe I am already warranted in making type identity claims concerning pain qualia (Baars, 2012; Churchland, 1989, 2002; Demertzi & Laureys, 2012; Schaffner, 2006). The missing details concerning pain appear to be just that: *missing*. Pain is mechanism.

5 A Mechanistic Description of Pain

In this section, I will delineate the physiological mechanisms that are type identical with pain qualia. This task involves elucidating how the experience of pain can be best described by specific interdependent nervous, endocrine and immune operations that together function as a single, comprehensive *mechanism*, of which tracts, systems and chemicals operate as component parts. Thus, I will refer to it as the nervous-endocrine-immune mechanism, or NEIM. I will delineate *coarse-grained* qualia of pain sensation (submodality, intensity, duration and location), pain negative emotion (unpleasantness), pain cognition (anticipation) and pain suppression (pain does not immediately follow wounding). In section 6, Table 1 summarises the three NEIM subsystems, their major component parts and sample operations. In section 6.1, Table 2 summarises the major chemical relationships between the NEIM subsystems.

5.1 Pain Submodality

Pain, a submodality of bodily sensation, like touch, proprioception and temperature, itself divides into further submodalities: thermal pain (burning skin on a hot surface), mechanical pain (crushing a thumb in a door), and chemical pain (lemon juice in an open wound). Nociception is the physiological operation by which thermal, mechanical or chemical stimuli are detected by a subpopulation of peripheral nerve fibres called *nociceptors* (e.g., Cesare & McNaughton, 1997; Schnitzler & Ploner, 2000; Willis, 1980; Zimmerman, 1976). Nociceptors also contribute to inflammation following wounding by releasing substance P (SP), calcitonin gene-related peptide (CGRP), neurokinin A (NKA), and nitric

oxide (NO). Nociception initiates neurogenic inflammatory operations that amplify responses to subsequent stimuli, whether noxious or innocuous.

The cell bodies of nociceptors are in the dorsal root ganglia (DRG) for the body (the trigeminal ganglion for the face), and have both a respective peripheral and central axonal branch that innervates their target organ and the spinal cord (Figure 1). Nociceptors are excited only when stimulus intensities reach noxious levels, indicating that they have physical properties that enable them to selectively detect and respond to potentially injurious stimuli. There are two types of nociceptors. One type of nociceptor includes small diameter unmyelinated 'C' fibres (Figure 1). C-fibres are activated by a variety of thermal, mechanical, and chemical stimuli and carry information from *polymodal* nociceptors. Roughly 70% of all nociceptors are C-fibres, but not all C-fibres are nociceptors. The other type includes medium diameter myelinated (Aδ) afferent neurons (Figure 1). These primary afferents differ from the larger diameter and rapidly conducting Aβ-fibres that respond to innocuous mechanical stimulation (i.e., light touch).

Figure 1: Nociceptors and the Fibres Carrying Pain to the Spinal Cord

Figure 1 Abbreviations: Aδ: Aδ-fibre; AST: anterior spino-thalamic tract (the paleospinothalamic tract); C: C-fibre; FNE: free nerve endings, primary afferent terminals in skin; DRG: primary afferent neurons in dorsal root ganglions; LST: lateral spino-thalamic

tract (the neospinothalamic tract); SN: primary afferent processes in spinal nerve; ST: spino-thalamic.

Aδ nociceptors are further subdivided into two main types (Figure 1). Type I (HTM: high threshold mechanical nociceptors) respond to both mechanical and chemical stimuli, but have relatively high heat thresholds (> 50 C°). If, however, the heat stimulus is maintained, these afferents will respond at lower temperatures. They will sensitize (i.e. the heat or mechanical threshold will drop) in the event of wounding. Type II Aδ nociceptors have a much lower heat threshold, but a very high mechanical threshold.

Nociceptors project to the dorsal horn of the spinal cord, which is organized into specific laminae (Schnitzler & Ploner, 2000) (Figure 1): C nociceptors project to laminae I and II (Figure 1, B); Aδ and Aβ nociceptors project to laminae I and V (Figure 1). Projection neurons within laminae I and V are the main output from the dorsal horn to the brain (Schnitzler & Ploner, 2000). These neurons are at the origin of several ascending anatomical tracts (pathways), including the neospinothalamic (Figure 2) and paleospinothalamic (Figure 3) tracts, which carry pain to S1, to THA and the brainstem, respectively. From there, information reaches cortical structures and regions; there is no single 'pain module' in the brain (Apkarian et al., 2005).

The qualitative difference between innocuous warmth and noxious heat enables us and other biological individuals to identify and avoid temperatures capable of wounding and causing pain. This pain threshold, which is approximately 43 C°, is *perfectly correlated* with the heat sensitivity of C and Type II Aδ nociceptors (Cesare & McNaughton, 1996). Capsaicin, the main pungent ingredient in 'hot' chilli peppers, and associated vanilloid compounds, produce burning pain by depolarizing subsets of C and Aδ nociceptors through activation of the capsaicin (or vanilloid) receptor, TRPV1; one of about 30 of the greater

transient receptor potential (TRP) ion channel family (Caterina et al., 1997; Caterina et al. 2000). Cloned TRPV1 channels are also gated by increases in ambient temperature, with a thermal activation threshold (~ 43 C°).

Ex-vivo skin nerve recordings have related mechanical stimuli to specific fibre subtypes. For example, Aβ-fibres are strongly correlated with sensitivity to light touch, whereas C and Aδ-fibres are primarily correlated with noxious mechanical stimuli. Pressure can be applied to the cell bodies of cultured somatosensory neurons using a glass probe, stretch through distension of an elastic culture surface, or changes in osmotic strength. Such studies show that pressure opens a mechanosensitive cation channel to cause rapid depolarization (Basbaum et al. 2009). The molecular basis of mammalian mechanotransduction is complex; ASIC (acid-sensitive ion) channels 1, 2 and 3 have emerged as the primary noxious mechanotransducers (Basbaum et al. 2009).

Chemo-nociception is nociception of environmental irritants and endogenous factors produced by physiological stress. In acute pain, chemo-nociceptive mechanisms trigger pain to a variety of environmental irritants: TRP channels function as receptors for plant-derived irritants, including TRPV1, menthol (TRPM8), as well as the pungent ingredients in garlic and mustard plants, isothiocyanates and thiosulfinates (TRPA1) (e.g., Caterina et al. 1997). Endogenous neural and non-neural substances including neurotransmitters (e.g., serotonin), peptides (e.g., bradykinins), lipids (e.g., prostaglandins, endocannabinoids) can activate chemo-nociception through binding to TRP receptors (Levine et al. 1993; Reichling & Levine, 1999). Some of these chemo-inflammatory nociceptors depolarize primary afferent neurons through iontropic mechanisms while others do so through metabotropic or second messengers.

I have argued that pain submodality is best described by nociceptor specialization. Each of the pain qualia (i.e., submodalities) is represented by nociceptors that are

submodality specific. When a nociceptor is stimulated naturally (e.g., skin burn) or experimentally (e.g., by electrical stimulation of the neuron), the quale experienced is specific to the information normally processed by the neuron (i.e., burnt skin). Accordingly, a 'mechanical' nociceptor (e.g., Aδ) will not fire to noxious heating of the skin or to a touch stimulus that does not noxiously compress the skin. The nociceptor and its connections in the CNS determine the submodality specificity of the neurons forming a somatosensory tract or pathway (e.g., neospinothalamic and paleospinothalamic tracts). Thus, nociceptor specificity best describes why different sensory qualia are different mechanistic submodalities.

5.2 Pain Intensity

A key feature of a noxious stressor such as wounding is pain intensity (McEwen, 2000, 2007; Selye, 1936). There is a robust correlation between the intensity of a noxious stimulus and verbal ratings using the Visual Analogue Scale (VAS) (Price et al. 1983; Price & Harkin, 1987). This relationship shows a positive power function with an exponent of 3.0-3.5 (Price and Harkin 1987). Facial reactions increase in intensity and frequency as a function of noxious stimulus intensity, and co-vary with verbal self-report of pain, when circumstances are suitable (e.g., Kunz et al. 2004). Pain facial expression can be intentionally controlled, but the upper face is less susceptible to voluntary control when pain is very intense (Rinn, 1984). Pain is often assessed by the degree of pain intensity. Different degrees of pain intensity are explained as Just Noticeable Differences (JND) (Hardy et al. 1947; Luce & Edwards, 1958). There are 22 JND for pain caused by heat to the skin. This differentiation is possible because the discharge frequency of the nociceptors increases with increasing skin temperature. JND is supported by many neural recording studies in nociceptors. These studies demonstrate that the operations in nociceptors parallel psychophysical evaluation of pain intensity for both noxious mechanical and thermal stimuli (e.g., Bromm et al. 1984; Gybels et

al. 1979). Thus, the level of the noxious stimulus presented and the intensity of the resulting pain quale is best described by the operations of nociceptors.

However, the descriptive relationship between stimulus intensity and signal transduction is not isomorphic, i.e., *one-to-one*. A complex ensemble of nervous, endocrine and immune mechanisms operates to modulate the eventual pattern of activity in nociceptors and pain intensity. Among these mechanisms, major component parts include second messenger substances such as CRH, potassium (K) (Jensen & Norup, 2002) and CORT (Bacigalupo et al. 1990) to influence receptors, the dorsal horn, which integrates *multimodal* sensory input from both external and internal environments, the hypothalamo-pituitary-adrenocortical axis (HPA), LC noradrenergic system (LCN) and the amygdala (AM), which interact with the hypothalamic periventricular nucleus (HPVN) to initiate the stress response (Chapman et al. 2008; Chapman, 2010).

5.3 Pain Duration

Acute superficial pain has a definite onset as well as offset; thus, it has duration. For example, facial displays of pain are visible as the response to acute clinical and experimental pain (e.g., Kunz et al. 2008), and diminish when analgesics are applied, in both normal (e.g., Taddio et al. 1997) and premature neonates (e.g., Scott et al. 1999). Some cutaneous wounds initially generate a *highly localized* 'first' pain that is followed by a *poorly localized* 'second' pain (e.g., stubbing a toe). This experience is called 'double pain sensation' (Campbell & LaMotte, 1983). First pain is described as lancinating, stabbing, or pricking; second pain is more pervasive and includes burning, throbbing, cramping, and aching and recruits sustained emotional components with descriptors such as 'sickening' (Price & Dubner, 1977a).

The temporal ordering of these specific pain qualia strongly correlates with nociception: firing of Type II Aδ nociceptors strongly correlates with the 'first' acute pain

response to noxious heat. Compression block of myelinated peripheral nerve fibres eliminates first, but not second, pain (Torebjörk & Hallin, 1973). The Type I fibre mediates the first pain stimulated by pinprick and other intense mechanical stimuli. The first of the double pain sensations reaches the CNS on the neospinothalamic tract (i.e., the LST) to the ventroposterolateral (VPL) and ventroposteromedial (VPM) nuclei of the THA and then to S1 (Willis & Westlund, 1997) (Figure 2). Alternately, a low dose of local anaesthesia applied to peripheral nerves blocks the C-fibres before the Aδ-fibres (Johansson et al. 1990). Under this condition, the slow conducting pain information is blocked (paleospinothalamic tract), and only the fast conducting pain information by Aδ-fibres carried to the CNS. The second of the double pain sensations reaches the CNS on the paleospinothalamic tract (i.e., the AST) to the brainstem nuclei and then to the parafasciculus (PF) and centromedian (CM) (i.e., the PF-CM mechanism) in the intralaminar THA (IL) (Willis & Westlund, 1997) (Figure 3).

The speed of noxious signal transmission following wounding is directly correlated to the diameter of nociceptor axons and whether or not they are myelinated. Aδ fibres have myelinated axons of around 2-6 μm in diameter with conduction velocities of 120-300 m/s. C fibres have unmyelinated axons of around 0.4-1.2 μm in diameter with conduction velocities of 5-20 m/s (Basbaum & Jessell, 2000). Just as nociceptor specificity best describes why pain has different submodalities, the distinct ‘first’ and ‘second’ pain qualia in double pain is best delineated by the differences in signal transmission in Aδ and C nociceptors, respectively.

Figure 2: The Neospinothalamic Tract and Pain

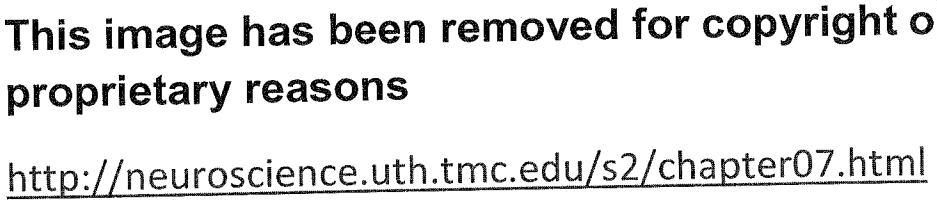
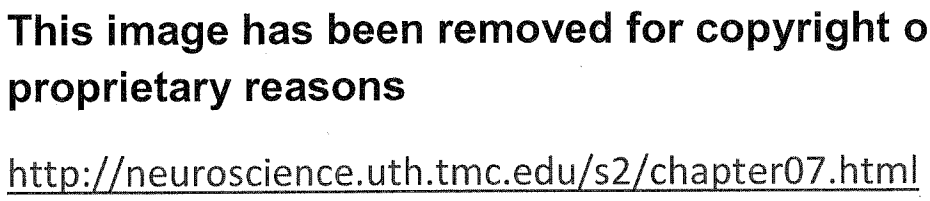


Figure 3: The Paleospinothalamic Tract and Pain



5.4 Pain Location

The response to a stressor such as a wound involves adaptive behaviours such as precisely locating the site of pain and mounting spatially oriented withdrawal actions (Chapman et al. 2008; Chapman, 2010, Melzack, 1999). Psychophysical studies have shown that the mean distance between the stimulus site of noxious cutaneous thermal stimuli and the perceived site (shown by pointing) was 10 mm on the palm, and 17 mm on the foot (Koltzenburg et al. 1993; Jorum et al. 1989; Ochoa & Torebjörk, 1989). Moore and Schady (1995) found that subjects could point out the site stimulated by noxious heat with a mean error of 14 mm. These findings suggest that selective activation of nociceptors encodes sufficient spatial information to identify locating a pain quale in a specific body part or location. This type identity claim requires neuroscientific descriptions of receptive fields and cortical somatotopy.

A receptive field is a region of sensory space that can produce neuronal responses when stimulated (Hubel & Weisel, 1959, 1962, 1968). In somatosensory neurons, sensory space is a 2-dimensional region of the skin or body and the stimulus can be pain. The receptive fields of Aδ nociceptors are small, and therefore allow precise localization of pain. The receptive fields of C nociceptors are large, and therefore allow less precise pain localization. Through line-labelling, spatially localized information reaches cortex where in primary somatosensory cortex (S1) and secondary somatosensory cortex (S2), there are several *somatotopic maps*.

Somatotopic maps are representations of the body surface identified with the post-central gyrus (PCG) of the parietal lobe (Jasper & Penfield, 1949; Penfield & Jasper, 1954) (Figure 4). Within the spinal cord, brain stem, THA and PCG, the location of a neuron is correlated with its receptive field. Accordingly, body and face (i.e., the receptive fields) are represented spatially (i.e., topographically) within nuclei and cortex such that neurons with

contiguous receptive fields are located next to one another within a specific structure. Adjoining areas of the body are represented in adjoining areas of the cortex. The resulting neural maps of the body and face are *not* isomorphic representations and appear distorted due to the disproportionate representation of the hand and face areas; that is, neurons representing the hand and face have small receptive fields. Because somatosensory neurons represent specific stimulus features and specific areas of the body or face, electrical stimulation of a restricted area of the PCG (e.g., the area representing the tongue) will produce a bodily (and not gustatory) quale that is felt as arising from the specific region of the body (i.e., the tongue) (Jasper & Penfield, 1949; Penfield & Jasper, 1954) (Figure 4).

Figure 4: The Somatotopic Representation of the Body and Face in the Postcentral Gyrus and Posterior Paracentral Lobule.

Figure 4 Abbreviations: INS: insula; LS: lateral sulcus; PCG: postcentral gyrus; PPL: posterior paracentral lobule; S2: secondary somatosensory cortex; VSC: viscerosensory

cortex.

The neospinothalamic tract (Figure 2) is type identified with the experience of precisely locating the site of pain (Treede et al. 1999; Treede et al. 2000). The cortical correlate of the spatial localization of painful stimuli is the S1[10]. Aδ-fibres terminate in the VPM, C-fibres terminate in PF-CM mechanism; all of these fibres are somatotopic. From there, they project axons that synapse on the S1. Animal studies demonstrate nociceptive afferents from lateral thalamic nuclei project into S1 (e.g., Gingold et al. 2005). Experimental results have shown somatotopic maps in S1 are perfectly correlated with pain loci.

The type identification of S1 with pain location was initially made by Head and Holmes (1911) based on observation of patients with parietal lesions. Hundreds of neuroimaging studies have revealed these somatotopic maps are correlated with pain loci by showing that noxious stimulation to different body regions (i.e., receptive fields) increase activity in specific regions of S1 that produce pain in a specific body region (e.g., Andersson et al. 1997; Apkarian et al. 2005; Bingel et al., 2004; Bushnell et al. 1999; Kanda et al. 1999; Ochoa & Torebjork, 1989; Ogino et al. 2005). Clinical observations show that focal parietal cortical lesions cause deficits of sensory-discriminative, but not of emotional dimensions of pain (e.g., Greenspan et al. 1999; Ploner et al. 1999). It appears that nociceptive spatial resolution is organized in a proximal-to-distal manner; that is, the spatial resolution of pain is finer in more distal than proximal body regions, similar to that of the touch system (Weissman et al. 2012).

[10] There is also evidence from neuroimaging studies that the INS is somatotopically involved in pain location (e.g., Henderson et al. 2007; Mazzola et al. 2009).

5.5 Pain Emotion

In addition to the somatosensory qualia of pain, pain is normally an unpleasant and stressful experience. The anguish over the possibility that pain will never end, as reported in many chronic pain patients, is identified in the suffering, despair and loss of hope observed in pain patients, spouses and family members (e.g., Cassell, 1991; Chapman & Gavrin, 1999; Kerssens et al. 2002; Stoll & Buchi, 2009; Tavoli et al. 2008).

The emotional aspects of pain are correlated with specific behavioural, anatomical and physiological mechanisms. Kunz et al. (2011b) used a well-established cognitive strategy (suggestions) to differentially modulate the somatosensory and emotional aspects of pain and found that suggestions for either increased pain unpleasantness or pain sensation caused specific changes in facial response patterns: facial movements of the nose, upper lip and also eyebrows were closely correlated with emotional aspects of pain, whereas facial movements around the eyes were closely correlated with somatosensory aspects.

Rainville et al. (1997) used suggestion to modulate pain emotion in subjects while their hands were submerged in hot water in order to maintain constant pain intensity. Subject self-reports showed that suggestion dissociated pain unpleasantness and intensity. Ploner et al. (1999) reported the case of a 57 year-old male who suffered a stroke that damaged the hand region of his right S1 and S2. Noxious cutaneous laser stimulation of his left foot caused a well-localized pain quale; however, when the same stimulus was applied to his left hand at three times the intensity it failed to cause pain. The patient "spontaneously described a 'clearly unpleasant' intensity dependent quale emerging from an ill-localized and extended area 'somewhere between the fingertips and shoulder' that he wanted to avoid" (Ploner et al. 1999: 213). Moreover, this patient was "completely unable to describe quality, localization and intensity of the perceived stimulus".

The operations of specific mechanisms in the paleospinothalamic tract is type identical with the emotional aspects of pain (Price, 2000) (Figure 3). The nerve cells that populate the paleospinothalamic tract are multireceptive or wide dynamic range nociceptors. Their axons cross and ascend in the spinal cord primarily in the AST. These fibres contain several tracts. Each of them synapses in three different locations: (1) In the mesencephalic reticular formation (MFR) and in the PAG (also termed the spinoreticular tract); (2) In the tectum (these fibers are also called the spinotectal tract); (3) In the PF-CM complex (also known as the spinothalamic tract).

From the PF-CM mechanism, these fibres synapse bilaterally in S2. There are extensive connections between the PF-CM, HYP and the limbic areas such as the ACC and the INS, which are involved in pain emotion in humans (e.g., Craig, 1996, 2002, 2003a, 2003b; Price, 2000; Rainville et al. 1997) and in nonhuman animals (e.g., Uhelski et al. 2012)[11]. The INS integrates the somatosensory input with the limbic components to produce the unpleasantness of pain (Vogt, 2005; Wiech & Tracey, 2009). The limbic structures, in turn, project to the HYP and initiate more *visceral* pain qualia (e.g., the sickness response). LCN projections extend widely throughout the limbic brain and can excite frontal-amygdalar circuits which are involved in negative emotion and the stress response. Frontal-amygdalar and frontal-PAG circuits may also modulate the intensity of pain unpleasantness (Likhtik et al. 2005; Tracey & Mantyh, 2007). Finally, circulating E released by the adrenal medulla following activation of the HPA contributes to the stress response, fear, and pain unpleasantness.

Craig et al. (1996) measured regional cerebral blood flow (rCBF) levels in a thermal grill illusion task. This is an illusion in which spatially alternating non-noxious warm and cool thermal stimuli leads to reports of pain unpleasantness despite neither stimuli producing

[11] These neural regions are also reliably activated during pain empathy in humans (Lamm et al. 2011; Morrison et al. 2004; Singer et al. 2004).

pain or unpleasantness when presented alone. Using a subtraction technique for the two conditions, Craig et al. (1996) found that only the increase in activity in the ACC explained the pain. Similarly, in the suggestion study conducted by Rainville et al. (1997), rCBF results following subtraction (i.e., hand in hot water) showed a significant increase in ACC activity that was strongly correlated with subject pain unpleasantness ratings. Finally, patients who have received bilateral anterior cingulotomy as a last resort for their debilitating chronic pain typically report post-surgery that they can still perceive somatosensory aspects of pain qualia following noxious stimulation, but they no longer feel it to be unpleasant (e.g., Foltz & White, 1962; White & Sweet, 1969; Wilkinson et al. 1999; Yen et al. 2005).

5.6 Pain Anticipation

The stress response involves the multisubsystem physiological response to wounding, and also the *anticipation* of wounding and pain in a specific body part (Chapman et al. 2008; Chapman, 2010; Melzack, 1999). In some situations, anticipation of pain may influence the immediate unpleasantness of pain (Price, 1999; Staub et al. 1971) and of non-noxious stimulation (Sawamoto et al. 2000). Pain anticipation appears to involve several qualia, such as cognitive appraisal, anxiety, systemic physiological arousal (e.g., increased heart rate and breathing), and distributed (i.e., diverged) attention from the source and body location of noxious input; the impact of these factors may vary based on the experimental instructions given to the subject and to past experience (e.g., Hsieh et al. 1999). Although anticipation of pain enables individuals to avoid bodily harm, for the chronic pain patient it becomes maladaptive and can lead to fear of movement, avoidance, and anxiety (Chapman et al. 2008; Chapman, 2010).

Distributed attention by continuous stimulus-evoked activity has been shown by electrophysiological experiments in the primary visual (e.g., Roelfsema et al. 1998) and

somatosensory cortex of the macaque monkey (e.g., Hyvarinen et al. 1980), and in human primary sensory areas (e.g., Brefczynski & DeYoe, 1999). Carlsson et al (2000) found increased contralateral somatotopic activation of S1 during anticipation and actual tickling of the foot, and decreased activation in S1 regions processing information from unattended parts of the body during anticipation of tickling (i.e., hands and face). Similarly, Drevets et al. (1995) and Porro et al. (2002) found increased contralateral somatotopic activation of S1 during anticipation and actual noxious and innocuous somatosensory stimulation to a foot, and decreased activation in nonsomatotopically related hand and face areas of S1. Using MEG, Worthen et al. (2011) found a continuous increase in S1 activity during the anticipation of pain (20-30 Hz), which continued during the pain phase but at a lower frequency (10-15 Hz). These findings support the conclusion that the somatotopic organization of S1 best describes the anticipation of pain in a specific body part.

5.7 Pain Suppression

Certain stressful or painful situations may produce analgesia. Soldiers wounded in battle (e.g., Beecher, 1946) or athletes injured in sports events (e.g., Silva et al. 2007) sometimes report that they do not feel pain during the battle or game. However, these individuals observe they feel pain after the battle or game has ended. Based on these anecdotes, researchers have hypothesized that the *stress* the soldiers and the athletes experienced *suppressed* the pain quale they later felt. This experience is called *stress induced analgesia* (SIA) (e.g., Butler & Finn, 2009; Willier et al. 1981). Many animal studies have also shown that noxious electrical stimuli can cause SIA (e.g., Amit et al. 1986; Maier et al. 1982). Rhudy and Meagher (2000) found that fear of an external stimulus can suppress pain in both humans and animals through activation of endogenous opioids, whereas anxiety

increases pain[12].

SIA is partly explained by endogenous pain control and opiate systems. Opioidergic neurotransmission is found throughout the CNS. Opioidergic neurotransmission influences nociception, and also homeostatic functions such as thermoregulation (Poddar, 1995), CRH, NE and cytokine mediated release of opioids following wounding (Rittner & Stein, 2005). High densities of opiate receptors are found in PAG, nucleus raphe magnus (NRM), in the spinal cord, HYP and HIPP. At the level of the spine, opiate receptors are located at the presynaptic ends of the nociceptors and at laminae IV to VII in the dorsal horn. Here, activation of opiate receptors produces *hyperpolarization* of the neurons, which inhibits the firing and the release of SP, thereby blocking pain signal transmission (e.g., De Koninck & Henry, 1991). Benedetti et al. (2013) found that when the meaning of the pain experience was changed from negative to positive through verbal suggestions, the specific opioid antagonist naltrexone and the cannabinoid antagonist rimonabant were co-activated and *increased* pain tolerance. However, the combined administration of naltrexone and rimonabant antagonized the increased tolerance. Benedetti et al. (2013) show that a positive approach to pain leading to pain reduction is identified with the co-activation of the opioid and cannabinoid systems.

The neural mechanism that comprises the PAG (in the upper brain stem), the LC, the NRM and the nucleus reticularis gigantocellularis (RGC) contributes to the descending pain suppression pathway (paleospinothalamic tract), which inhibits noxious sensory signalling at the spinal cord level. For example, Fields and Anderson (1978) found that morphine administered in the PAG and NRM produces analgesia. It was discovered that noxious

[12] There is evidence that physicians may suppress some pain emotion in response to a patient's pain. For example, Cheng et al. (2007) found that the neural response of acupuncturists who believed acupuncture not to be painful was different than that of naïve participants to this practice. Whereas the latter group showed the expected pain-related response including activation in the AI, ACC, and S1 and S2, the former group did not. The physicians also showed additional activations in the medial and superior prefrontal cortices and the temporoparietal junction, two neural areas known to be correlated with emotion regulation and the ability to make inferences about mental states such as emotions, desires, and beliefs. This suggests that, with experience and perhaps with disbelief (or discounting of pain in others), physicians might be suppressing part of the spontaneous response to the pain of others.

stimulation activates the RGC. In turn, the RGC innervates both the PAG and NRM. The PAG sends axons to NRM, and neurons in NRM send their axons to the spinal cord. Moreover, bilateral dorsolateral funiculus (DLF) lesions block the analgesia produced by both electrical stimulation and by microinjection of opiates directly into the PAG and NRM, but they only weaken the systemic analgesic effects of opiates. These findings led to the type identity claim that activation of specific descending pathways in the DLF is opiate-mediated SIA (e.g., Abbott et al. 1996; Basbaum et al. 1976; Fields & Anderson, 1978; Lewis et al. 1983; Maier et al. 1982).

6 The Nervous-Endocrine-Immune Mechanism (NEIM)

I conclude that somatosensory qualia of pain sensation including submodality, intensity, duration and location, are best described by the operations of multisubsystem mechanisms in the neospinothalamic tract; negative emotional pain qualia by the operations of multisubsystem mechanisms in the paleospinothalamic tract; pain anticipation by the somatotopic organization of the primary somatosensory cortex (neospinothalamic tract); and pain suppression by the operations of the dorsolateral funiculus pathway and opiate systems within the paleospinothalamic tract. The experience of pain is best delineated by interdependent and integrated operations in nervous, endocrine and immune physiological systems. The major component parts of the NEIM are the neospinothalamic and paleospinothalamic tracts, the parts of the nervous, endocrine and immune systems as well as endogenous neurotransmitters, cytokines, hormones, peptides, and endocannabinoids. The primary operations of the NEIM are transduction and diffusion (nervous system), circulation (endocrine system), and migration (immune system). Systemic circulation is also involved,

often to sustain feedback loops[13]. The complex interdependency of these parts enables an adaptive, organized, multilevel response to the stressor of wounding followed by pain (cf. Blalock, 1994). The NEIM nests with a larger system that I roughly characterize as the *biological individual.* I claim that the functioning NEIM is type identical to the personal experience of pain. Table 2 summarises the three NEIM subsystems, their major parts and sample operations type identified with pain.

Table 2: Systems, Major Parts and Sample Operations of the Nervous-Endocrine-Immune Mechanism (NEIM) Type Identified with Pain

NEIM System	Major Parts	Major Operations
Nervous	▪ Aδ and C nociceptors. ▪ CGRP, P, NKA, NO. ▪ Dorsal horn (spinal cord). ▪ ACC, AM, PF-CM, DLF, HPP, HYP, INS, PAG, LC, SC, S1, S2, THA.	▪ Detects threat in the external environment. ▪ Signals wounding. ▪ Mounts 'fight or flight'. ▪ Cognition and emotion.
Endocrine	▪ HPA, HPNV, LCN, SAM. ▪ ACH, CORT, CRH, E, NE. ▪ CRH-1, CRH-2.	▪ Initiates 'fast' arousal immediately following wounding. ▪ Initiates 'slow' recovery, behavioural adaptation and return to normalcy.
Immune	▪ Proinflammatory cytokines IFN-γ, IL-1, IL1-β, IL-6, IL-8, TNF-α.	▪ Detects microbial invasion and toxins. ▪ Initiates inflammatory responses.

1. [13] Systemic circulation is the general circulation of the blood through the body, as opposed to the circulation of the blood from the heart to the lungs and back to the heart.

Three falsifiable claims are entailed by this theory; namely, the NEIM shows: (1) connectivity; (2) cross-subsystem feedback loops; (3) dysregulation within one or more subsystem leads to chronic pain. I will discuss these claims each in turn.

6.1 Chemical Connectivity

The chemical connectivity of the parts of the NEIM is extraordinarily complex. Many of the major parts of the NEIM are *pleiotropic*. That is, whether a specific effect (i.e., excitation or inhibition; arousal or recovery; pro-inflammation or anti-inflammation) is produced by a part or parts, or a specific quale, typically relies on mechanism context. For example, CRH performs several multilevel functions across multiple physiological systems, and can exert both pro- and anti-inflammatory effects, depending on its location and role (e.g., Merali et al. 2003; Merali et al. 2004; Müller et al. 2004). Table 3 presents a sample of chemical interactions of major parts of the NEIM type identified with pain.

Table 3: Examples of Chemical Connectivity of Major Parts of the Nervous-Endocrine-Immune Mechanism (NEIM) Type Identified with Pain

NEIM Subsystems	NEIM Subsystem Interactions Type Identified with Pain
Nervous-Endocrine	▪ The hypothalamic PVN and LC initiate hormonal responses and provide the mechanisms of feedback-controlled regulation. ▪ Hormones such as CRH and NE act as neurotransmitters in the nervous system and as endocrine hormones.
Nervous-Immune	▪ Microglia, oligodendrocytes, and astrocytes contribute to inflammation and feedback loops. ▪ C-fibres release CHR into a wound. ▪ The endocannabinoids endogenous ligands anandamide (AEA) is involved in chemical nociception. ▪ The sensory vagus and glossopharyngeal nerves have paraganglia that detect peripheral proinflammatory cytokine release. ▪ Proinflammatory cytokines synthesize cytokines within the CNS at microglia.
Endocrine-Immune	▪ Glucocorticoids inhibit proinflammatory (Th1) cytokine production to protect against overshoot in the inflammatory response. ▪ CRH acts as a releasing factor for endogenous opioids and cytokines by regulating local inflammation following wounding. ▪ Endocrine-Immune interactions often exploit systemic circulation.

The impact of the major NEIM parts does not reduce to specific operations they perform at a specific physiological level. That is, chemical connectivity is not to be type identified with mechanical operations at the *molecular* and *cellular* level, as *ruthless reductionism* claims (e.g., Bickle, 1998; 2003; 2006). Instead, connectivity in the NEIM is best described in terms of *mechanistic reductionism* that type identifies pain in terms of chemicals integrated into multilevel *systems* and neuroanatomical tracts (e.g., Bechtel, 2009). At the systems level, the parts of the NEIM describe numerous functions: communicate

information to enable ongoing subsystem coordination; contribute to negative or positive feedback loops that facilitate or delay homeostasis, and enable allostasis during stress; navigate the physical and social environment; adapt to wounding in real time, prepare and execute adaptive responses, and recover from those responses.

6.2 Feedback Loops

Contemporary pain science focuses on delineating the physiological mechanisms to best describe the experience of pain (e.g., Bonica, 1953; Melzack & Casey, 1968; Melzack & Wall, 1988; Perl, 2011). Historically, the scientific and humanistic project of understanding pain in terms of mechanism typically proceeded on the simple assumption that the target mechanism had a specific beginning condition and continued its operations until the finish or termination conditions are achieved (Perl, 2011). This *sequential* conception of mechanism is most clearly conveyed in the description proposed by Machamer et al. (2000): 'Mechanisms are entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions.' If the set-up (start) conditions involve a stimulus external to the mechanism, a mechanism is not only *sequential* but also *reactive* (Bechtel & Abrahamsen, 2010).

This reactive conception of a mechanism agrees well with the description of pain offered in science and humanities since Descartes (1637, 1664) and before (Perl, 2011), but it is insufficient to delineate the *endogenously active* features of physiological mechanism (e.g., Northrop, 1999). That is, some of the operations of the NEIM are *internally* generated; the causes and regulation of these operations is *inside* the NEIM rather than reactive to inputs external to the mechanism. A sequentially organized mechanism will not reveal endogenous operations (Bechtel & Abrahamsen, 2010; Northrop, 1999). Minimally, a physiological mechanism capable of endogenous operations preserves the sequential idea of the overall

functioning of the mechanism but permits operations that are viewed as *later* in the sequential order to *feed back*, either negatively or positively, on operations thought of as *earlier* (Franklin et al. 1994; Jones, 1994; Northrop, 1999). Adopting these organizational concepts entails delineating mechanism in terms of parts, operations and *dynamics*: "A mechanism is a structure performing a function in virtue of its component parts, component operations, and their organization. The orchestrated functioning of the mechanism, manifested in patterns of change over time in properties of its parts and operations, is responsible for one or more phenomena" (Bechtel & Abrahamsen, 2010).

Dynamic feedback operations cross nervous, endocrine, and immune systems and contribute to overall functioning of the NEIM. For example, cross-subsystem feedback loops facilitate the interdependence of endocrine and immune systems (e.g., Benarroch, 2006; Rivest, 2001). Glucocorticoid products of the HPA influence the operations of cytokine-producing immune cells which, in turn, influence the operations of the HPA. These hormones protect against overshoot in the proinflammatory response to the stressor of wounding (Chapman et al. 2008; Chapman, 2010; Melzack, 1999).

To simplify, negative feedback allows stability while positive feedback allows instability (Ferrell, 2002; Franklin et al. 1994; Jones, 1973; Northrop, 1999). In the event of wounding and subsequent pain, negative feedback protects homeostasis and minimizes allostatic load (see section 7.4); positive feedback induces defensive arousal and emergency behaviours (Chapman et al. 2008; Chapman, 2010; Melzack, 1999). A positive feedback loop is typically controlled by a negative feedback system that regulates overshoot and can eventually terminate the positive feedback loop (Ferrell, 2002; Franklin et al. 1994; Jones, 1973; Northrop, 1999). Concerning pain, feedback loops are adaptive in the short-term (Chapman et al. 2008; Chapman, 2010; Melzack, 1999). Periods of sustained positive feedback can be destructive. For example, chronic noxious input to the dorsal horn can

increase glutamate to toxic levels and thereby destroy inhibitory interneurons. Such damage is revealed in the formation of *dark neurons* (Hassanzadeh & Ahmadiani, 2006). Thus, chronic inflammatory noxious signaling can cause dorsal horn pathology. Opioid medications provide a strong example, as they resemble β-endorphin and other endogenous opioids. The HPA responds to such substances as though they were endogenous signals and the result can be hypogonadism (e.g., Daniell et al. 2006).

Negative and positive feedback can dysregulate the operations of the NEIM. The effect is a disease process (Chapman, 2010). Negative feedback may become dysregulated when an endogenous chemical allowing feedback disappears, occurs in excess, or becomes misidentified by exogenous products that are structurally similar. For example, opioid medications in a male pain patient dysregulated the HPG and produced hypogonadism (e.g., Bliesener et al. 2005). Dysregulation of chronic, stressor-related positive feedback likely contributes to allodynia, severe idiopathic abdominal pain, migraine headache, and many multisymptom disorders (Chapman, 2010; Melzack, 1999).

6.3 Dysregulation and Chronic Pain

Dysregulation is chronic dysfunction in a mechanism to recover its normal operations and interaction with other mechanisms following a stressor. It localises to any level of mechanism, whether it is the HPA (Blackburn-Munro & Blackburn-Munro, 2003; Kloet, 2006), or a biological individual in the social environment (van Rysewyk, 2009, 2010). I claim that pain becomes chronic and disabling following regulatory problems developing over time within the NEIM. Dysfunctional operations in one NEIM subsystem will likely result in dysfunction in the other subsystems because, as I have emphasized, they function reciprocally. Chronic dysregulation can cause irreversible organ disorder, which can effect noxious signaling, as in rheumatoid arthritis (Zautra et al. 2007). In chronic pain patients,

dysregulation may present in several ways (Chapman et al. 2008; Chapman, 2010; Melzack, 1999). I will briefly outline two presentations: incomplete recovery, and abnormal cross-system coordination.

Following wounding, recovery of the NEIM may occur, but fail to return the NEIM to normal functioning. For example, incomplete recovery in the HPA can result either in CORT deficiency, leading to chronic anabolism; or CORT excess, leading to chronic catabolism (Garofalo et al. 2007; Gatchel et al. 2006; Kloet, 2006). In both cases, abnormal diurnal variation in CORT pulsing indicates dysregulation. Thus, a dysfunctional endocrine recovery process is a mechanism for chronic endocrine dysregulation with multiple chronic symptoms.

Normally, acute dorsal horn facilitation subsides with wound healing. The dorsal horn is a multisynaptic structure that produces spinal reflexes, communicates nociceptive input to higher CNS structures, and modulates to either inhibit or facilitate nociceptive transmission, depending on information from higher CNS structures or from the periphery (Willis, 1985). Changes at the spinal cord level can alter higher CNS system functioning (central sensitization) and contribute to chronic pain. For example, sensitization at the dorsal horn results in long-term potentiation at hippocampal and cortical levels, and expansion of the receptive fields of nociceptors to include noninjured areas near the wound and normally innocuous sensory signals (Ji et al. 2003). Dysfunction in the activation of N-methyl-D-aspartate (NMDA) receptors via glutamate, the main CNS excitatory neurotransmitter, is a source of dorsal horn dysregulation following wound healing (e.g., Dubner, 2004; Millan, 1999).

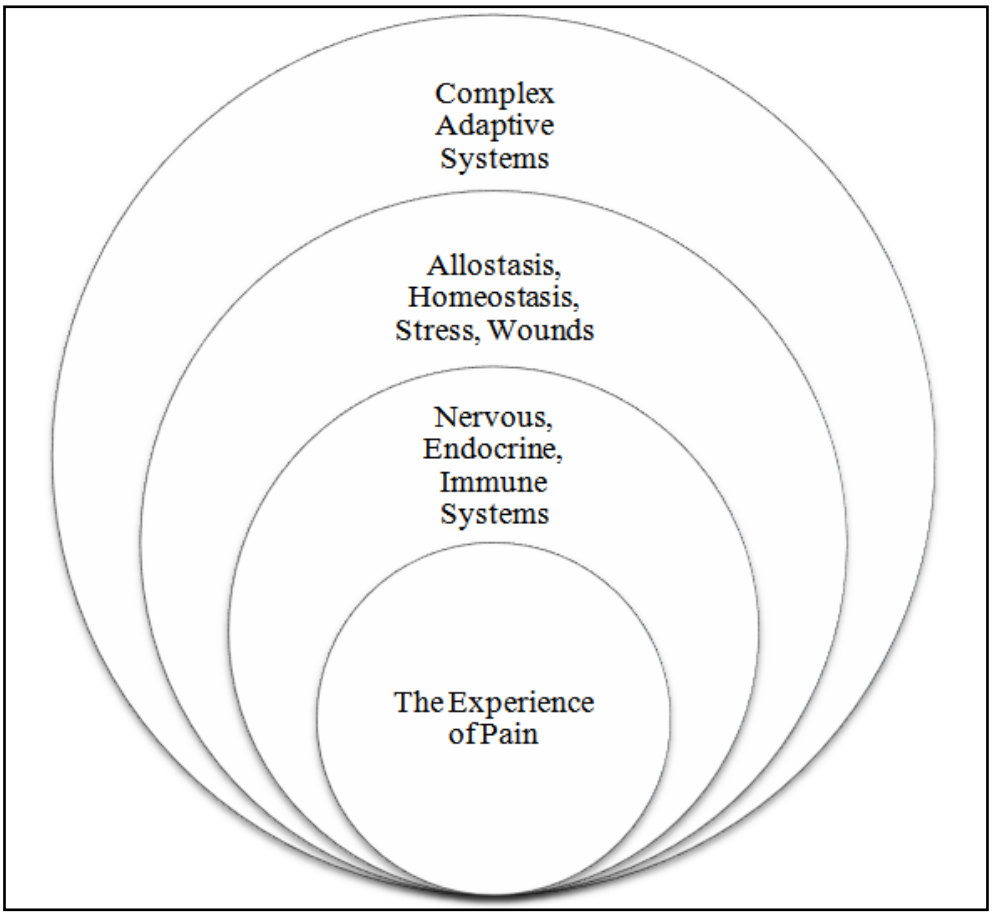
The connectivity required for cross-subsystem coordination in the NEIM may also fail. Examples relevant to chronic pain include chronic dysfunction between cytokine regulation and LC activity (e.g., Borsody & Weiss, 2002), cytokine regulation and HPA regulation (e.g.,

Blackburn-Munro & Blackburn-Munro, 2003; Calcagni & Elenkov, 2006; Rivest, 2001), and cytokine regulation and autonomic regulation (Czura & Tracey, 2005).

7 Fundamental Framework Concepts

The delineation of correlations between pain qualia and physiological mechanism leads to the type identification of pain with the NEIM. Although empirical progress in the understanding of pain is typically gradual and piecemeal (Perl, 2011), the type identification of pain qualia with the NEIM does not proceed in an additive manner. Pain scientists do not discover one pain type identity at a time and then add them together (McCauley & Bechtel, 2001). Rather, what justifies descriptive claims to have type identified the mechanisms of pain is the way the entire multilevel mechanistic package *coheres*. The ultimate ambition of this project is to provide a coherent account that best describes pain, and – in the next chapter – best explains pain facial expression. While the different features of pain are best described by different component parts and operations of the NEIM, as I argued in section 5, it is relevant to note with regard to this point that the major parts and operations of the NEIM are *already described and consilient with well established scientific descriptions*. At the same time, just like interdependence in the functioning components of the NEIM, no one of the pain type identifications is truly descriptive without the others, nor without a prior understanding of more basic descriptive concepts necessary to understanding the nature of pain. Hence, in this section, I will delineate the fundamental concepts that best describe type identifying pain with the NEIM. To satisfy this requirement, I offer a multilevel, conceptual description of the following: homeostasis, allostasis, stress, CAS, and wounds. The interdependent descriptive relationship between these concepts and the personal experience of pain is shown in Figure 5.

Figure 5: The Relationship between Complex Adaptive Systems, Allostasis, Homeostasis, Stress, Wounds, Physiological Systems, and the Experience of Pain



A concise description of the entire philosophy on offer in this project, including the core type pain type identity claim, NEIM, and CAS theory, is this:

A biological individual is a complex adaptive system coping with a physical and social environment, but possessing nested subsystems. Pain is an allostatic stress mechanism comprised of interdependent nervous, endocrine and immune operations.

7.1 Homeostasis

Homeostasis maintains the necessary functions of the internal environment such as blood pressure, blood gases, fluid levels, metabolite levels, thermoregulation, and acid base (Cannon, 1929; McEwen, 2000, 2007; McEwen & Wingfield, 2003). Failure to sustain homeostasis is *fatal*. The NEIM contributes to homeostasis in the event of wounding or injury (e.g., Goetzl & Sreedharan, 1992; O'Connor et al. 2004; Wilder, 1995). Homeostasis is threatened by environmental extremes, depletion of essential resources, extreme physical exertion, abnormal feedback processes, wounding, disease, and aging. In response to a stressor that exceeds a threshold magnitude, a biological individual alters its behaviour and physiology to sustain homeostasis. Thus, homeostasis is self-limiting and uses *negative feedback loops* to return these physiological parameters to a resting state. Homeostasis does not assist in adaptation; instead, adaptation facilitates homeostasis (McEwen, 2000, 2007; McEwen & Wingfield, 2003).

7.2 Allostasis

The interdependent physiological and adaptive response serving homeostasis is *allostasis* (McEwen, 2000, 2007; McEwen & Wingfield, 2003). Allostasis ensures that the operations maintaining homeostasis stay within normal range. That is, homeostasis keeps a biological individual *alive* while allostasis enables that individual to *adapt*. Allostasis allows the body to produce hormones (e.g., CORT, CRH, E, NE) and other mediators (e.g., CGRP, SP, cytokines) that assist an individual to adapt to novel or threatening events, such as injury and pain. Thus, allostasis is the core of the stress response because it recruits internal resources to meet the challenge of a stressor. When a stressor, such as a wound, persists for an extended period of time, or when the mediators of adaptation (e.g., CORT) occur chronically, allostasis may mobilize resources faster than the body can restore them. A

subsystem regulated by negative feedback may fail through (e.g.) depletion of a key neurotransmitter or peptide (Staines, 2006). That is, the allostatic load causes dysregulation. *Allostatic load* designates the cost of allostatic adjustment to the body (McEwen, 2000, 2007).

The psychosocial system in which an individual is nested can initiate allostasis and lead to chronic pain even in the absence of wounding or pathology (e.g., Blackburn-Munro & Blackburn-Munro, 2003; Heim et al. 1998; Wingenfeld et al. 2008; Wingenfeld et al. 2009). The relationship between stress history, psychopathology and HPA changes in women with chronic pelvic pain (CPP) is a case in point. While diagnostic laparoscopy typically shows a normal pelvis in many patients with CPP, psychological studies show a high frequency of psychopathology and increased incidence of chronic stress and traumatic life events (i.e., sexual and physical abuse) in women with CPP, indicating a relationship between post-traumatic stress disorder (PTSD) and CPP. Heim et al. (1998) found that women with CPP had alterations in the HPA, that partly associated with neuroendocrine correlates of PTSD, and that were similar to findings in patients with other stress-related bodily disorders. Heim et al. (1998) suggested that a CORT deficiency partly describes the development of bodily disorders in chronically stressed or traumatized individuals.

7.3 Stress

Stress is preparation and execution of allostasis in response to novel or threatening stimuli in the internal or external environment (Johnson et al. 1992). A *stressor* is any stimulus or quale that causes a *stress response*. It may be a sensory signal of wounding (Blackburn-Munro & Blackburn-Munro, 2003; Melzack, 1999), a social event such as observing another individual in pain (van Rysewyk, 2009, 2010), an invading micro-organism, or starvation. Thus, stressors can be positive (adaptive, rewarding) or negative (threatening, harmful). The main explanatory features of a stressor are intensity, duration and

frequency (Selye, 1936). Selye (1936) identified the stress response as having three stages: alarm, resistance, and exhaustion (if a stressor persists). A normal stress response involves alarm, resistance and recovery. Studies on humans and in animal models show that the context in which the physiological responses occur can influence the intensity and duration of the stress response (e.g., Hüther, 1996; Johnson et al. 1992; Strange et al. 2000). Thus, psychosocial factors can type identify whether a stimulus or quale is a stressor. Cognitions such as beliefs, expectations, and attributions are also stressors (cf. sections 5.6 and 5.7). While sensory noxious signalling normally causes the stress response, negative thinking can be a stressor and influence pain, as in *pain catastrophizing*, which is predictive of pain intensity and related disability (e.g., Severeijns et al. 2001; Sullivan et al. 1995).

7.4 Complex Adaptive Systems

A living biological individual is a complex, open, adaptive system (CAS) that follows the twin evolutionary objectives of survival and adaptation to the environment (e.g., Dooley, 1997; Holland, 1992; Kaneko, 2006; Lansing, 2003). The term *system* refers to a class of components parts comprising a *whole* within which each component interacts with at least one other component. All system components fulfil a common objective. Every CAS contains nested subsystems that function as component parts. Nervous, endocrine, and immune systems are among the functionally interdependent systems that comprise the body.

A CAS has three essential features. The first essential feature is *irritability*. The system is dynamic and reacts to disturbances such as wounding by moving away from allostasis to cope with the challenge and returning toward allostasis afterward. The second essential feature is *connectivity*. Connections and interactions exist among the components parts of a system. Connectivity enables pattern formation and self-regulating positive and negative feedback. As a result, the connectivity of a system is more important than the system components

themselves. The third essential feature of a CAS is *plasticity*. A CAS operates selectively in reaction to changes in the environment, and change is usually nonlinear. An essential feature of system nonlinearity is that small disturbances can yield large system changes (i.e., sensitization) while large disturbances sometimes do not. For example, nonlinear shifts in the normal functioning of the CNS to states of either facilitation or inhibition can change somatosensory thresholds to normally non-noxious stimuli to produce noxious signaling (e.g., *mechanical allodynia*) or to stimuli that normally would have produced minimal pain can become intensely painful (*hyperalgesia*) (Chapman et al. 2008; Chapman, 2010).

It has long been known that pain serves biological functions (Perl, 2011). Escape and avoidance of physically dangerous events enhances survival and reproduction. Pain alerts the individual to the presence of a potentially harmful stimulus in the environment, initiates withdrawal behaviours, promotes healing following injury by limiting movement, and facilitates avoidance of such stimuli in future encounters. This is illustrated by individuals who have congenital abnormalities such as CIP that render them incapable of detecting painful stimuli (e.g., Berthier et al. 2004; Cox et al. 2006; Rubins & Friedman, 1948; Schilder & Stengel, 1931). These patients cannot feel piercing pain from a sharp object, heat of an open flame, or even discomfort associated with internal injuries, such as a broken bone. Consequently, they do not engage appropriate protective behaviours against these conditions, many of which can be life threatening. However, not all pains are adaptive. Dysregulation of the NEIM can result in chronic pain *and* stress-related disorders such as anxiety and depression (Melzack, 1999; Raison, 2009; Wingfield et al. 2009). Chronic pain may be a side-effect of plasticity (Chapman, 2010; Melzack, 1999). Thus, the impact of a wound can extend beyond its local tissue environment to its interactions with higher CNS systems (e.g., Grande et al. 2004; Wingenfeld et al. 2008).

7.5 Wounds

A wound is a disturbance of normal anatomical structure and function (Lazarus et al. 2002; Sonnemann & Bement, 2011; Young & McNaught, 2011). Wounds result from pathologic operations that commence externally or internally, and arise in accidental or intentional trauma or disease. They are typically acute, but may become chronic. In acute wounds, healing is complete and organized; in chronic wounds, incomplete and disorganized. A chronic wound may remain indefinitely (Velnar et al. 2009). Painfulness and personal distress can increase sympathetic activity systemically through autonomic and endocrine mechanisms, and this may hinder normal wound healing by reducing blood flow (Sonnemann & Bement, 2011; Young & McNaught, 2011). Cutaneous, musculoskeletal and visceral wounds may fail to heal following injury, continuing as disorganized, locally inflamed operations that respond maladaptively to changes in the NEIM. Chronic pain may result (Chapman, 2010; Melzack, 1999).

Injury disturbs the local tissue environment, causes inflammation and coagulation, constricts blood vessels, and initiates immune operations (Lazarus et al. 2002; Sonnemann & Bement, 2011; Young & McNaught, 2011). Sympathetic operations at the wound restrict blood flow. Vasoconstriction immediately following injury blanches the wound and lessens hemorrhage, fosters platelet aggregation, and maintains healing factors within the wound. Next, vasodilation produces edema and heat. C-fibres interact with wounds, by releasing proinflammatory peptides such as SP, CGRP, and NKA and by signaling injury. Proinflammatory cytokines IL-1, IL-6, IL-8, and TNF-α, macrophages, neutrophils, and proteins produce a systemic *acute phase response* that safeguards against microbial invasion, and sensitizes the wound to facilitate healing (e.g., Tracey, 2002). An acute wound is a multitude of interdependent cellular and molecular operations that initially restore the immune barrier disturbed by tissue trauma injury and then repair lost normal tissue structure

(Velnar et al. 2009).

8 Conclusions

This chapter aims to offer the best descriptive answer to the mind-body puzzle: *what* is pain? I have described the physiological mechanisms that describe pain qualia within the context of advancing theoretical descriptions of the nervous, endocrine and immune systems and their reciprocal relationships (i.e., the NEIM), descriptions of allostasis, homeostasis, stress and wounds, and complex adaptive system-based descriptions of pain. These delineations collectively best show how pain qualia are type identified with neurophysiological mechanism. My descriptive account type identifies somatosensory pain qualia (modality, intensity, duration and location) with multisubsystem and multilevel operations of the neospinothalamic tract; negative emotional pain qualia with operations of the paleospinothalamic tract; cognitive pain qualia (pain anticipation) by the somatotopic organization of the primary somatosensory cortex (neospinothalamic tract); and pain suppression (stress-induced analgesia) by the dorsolateral funiculus pathway and opiate systems within the paleospinothalamic tract. These type identities are supported by general descriptions of the nervous, endocrine and immune systems, and understanding of allostasis, homeostasis, stress and wounding. The entire package is constrained by the fundamental assumption that biological individuals are complex adaptive systems. Thus, the simplest and most tractable descriptive metaphysic of these robust relationships is that pain *is* mechanism.

The type identity theory of mind is traditionally proposed as a metaphysical philosophy of sensations, or conscious mental states generally. Whether a type identity theory can best describe the phenomena of intentionality and embodiment in the experience of pain has not been previously investigated. I suggest that future type identity theories of mind be advanced to describe type identities between pain and specific types of embodied behaviours,

such as self-care during personal pain, and between pain and specific types of observer experiences, such as empathy, pain empathy synesthesia, or indifference leading to exploitation of the one in pain. In the next chapter, I attempt to meet the theoretical challenge of providing a type identity theory of pain embodiment. I propose an original hybrid polyvagal and type identity theory of pain facial expression in order to best explain the following *explanatory* mind-body puzzle: *how* can pain exist?

3 Bridging the Explanatory Mind-Body Gap: A Polyvagal-Type Identity Theory of Pain Facial Expression

Abstract

In this chapter, I advance a novel polyvagal-type identity theory of pain facial expression to best explain the *explanatory* mind-body puzzle: *how* can pain exist? According to some philosophers, assuming neuroscience explains with what mechanistic operations being aware of a burning arm pain is type identical, it is still impossible for any neuroscientific theory to explain how a specific pain must be correlated with a specific mechanism, as opposed to a different mechanism. Thus, there appears to be an explanatory gap. In this chapter, I will attempt to bridge the explanatory gap in two ways. First, based on the theoretical approach for a type identity theory of pain offered in Chapter Two, I offer an original polyvagal-type identity theory of pain facial expression. I claim type identity theory of mind best explains how the gap can be bridged. Type identity theory of mind makes a realist assumption that pain is causally responsible for behaviours such as facial pain grimaces and screaming. Second, I attempt to bridge the gap by arguing that the supposed gap assumes that type identity theory must reconstruct type pain identities as formal derivations from laws of nature. Based on actual scientific practice and philosophical considerations concerning explanatory levels, I will show that this is a false assumption. Type identity statements that successfully emerge from mechanistic pain explanation are between different delineations of pain phenomena at the *same* explanatory level; they are intralevel. Although the explanatory gap puzzle correctly shows our incomplete understanding of how pain might be explained by mechanism, the gap merely asserts a practical limit on our present explanatory successes, and is not in principle unbridgeable, as some philosophers assert.

Keywords: deductive-nomological model, diagrams, discovery, explanatory gap, explanatory level, generalization, mechanism, mechanistic explanation, pain, pain facial expression, polyvagal theory, type identity theory of mind

1 The Explanatory Gap Problem

The ambition of this project is to make the case for a type identity theory of pain. The core claim of the theory on offer is that every member of the type *pain qualia* is necessarily a member of the type *operations of the NEIM*; that is, the two types are identical. Some philosophers think that facts about physiological mechanism cannot decide questions about conscious pain because pain is of a completely *different type* (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Levine, 1983, 2001; McGinn, 1991; Ray et al. 2013). Advocates of this philosophy have argued that qualia inversion is physiologically undetectable, that neuroscience excludes pain, or makes it disappear, that an exact physical copy of a person could have different pains than the original person, or could have no pains at all. These philosophers think our ability to imagine these scenarios reveals deep *metaphysical* truths about pain, mind and consciousness. In Chapter One, I offered many criticisms of these arguments.

However, these same philosophers and scientists have found that type identity theory faces an additional philosophical problem; namely, *how* can pain exist? Assuming neuroscience discovers with what mechanism being conscious of a pain in one's left thigh is type identical, we would still not understand how and why precisely *this* mechanism is type identical with just *that* pain quale, rather than (e.g.) a quale of pleasure (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Levine, 1983, 2001; McGinn, 1991; Ray et al. 2013). Neuroscience, the claim goes, cannot ever explain how and why a specific pain quale is type identical to a specific mechanism, as opposed to a *different* mechanism, and how and why a specific mechanism is not type identical with a *different* type of quale altogether. The next step in the objection may be to conclude that we cannot ever hope to explain type pain identity claims in terms of neuroscience. Thus, neuroscience will never be able to explain the

conscious experience of pain. In the words of Levine (1983, 2001), there is an *explanatory gap*.

The explanatory gap makes three claims. One, the gap does not deny the many empirical correlations between pain and mechanism discovered by neuroscience. Such correlations are *real*. The problem neuroscience faces concerning the gap is that the correlations it has found characterize relationships that are only *contingently* true, true as a matter of fact, *not true as a matter of necessity*. To bridge the explanatory gap, neuroscience needs to demonstrate that there are correlations between pain and mechanism, and that these relationships are *necessary*. Two, the gap does not deny that type identifying conscious pain with mechanism will bridge the gap. This is because type identity is a *necessary* relation. Thus, genuine neuroscientific correlations between pain and mechanism can bridge the gap if they are type identified. However, many philosophers and some neuroscientists believe type identity theory of mind is false (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; McGinn, 1991; Ray et al. 2013). Last, even if type identity theory is true, it still needs to explain how and why a particular pain is type identical to a specific mechanism, rather than a different mechanism, or conversely, how and why a specific mechanism is not type identical with an entirely different type of experience. Many philosophers believe type identity theory cannot offer such explanations. I challenged this belief in Chapter One and Chapter Two.

Philosophers and scientists have offered various responses to the alleged explanatory gap. Searle (1992) acknowledges the reality of the gap, but denies that it contradicts a physicalist view of experience, because only some physical qualities are irreducibly subjective. Other philosophers suggest that the gap may be *bridged* in the future, but presently we lack the concepts to unify personal experience and mechanism. For instance, Nagel (1974) thinks qualia may prove to be physiological, but we currently lack any clear

idea as to how they *could* be. McGinn (1991) feels that the gap is *unbridgeable* by us or by any creatures like us. The concepts we have and the concepts we are capable of forming forever closes us to a complete, bridging explanation. Other philosophers and scientists agree that the gap is unbridgeable, but go on to claim that the correct conclusion to infer is that there is a parallel *metaphysical gap* in the *world*. Conscious experiences like pain have irreducibly subjective, nonphysical qualia (e.g., Chalmers, 1996; Derbyshire & Raja, 2011; Feinberg, 2012; Ray et al. 2013). This last view should be familiar, as it is the dualistic philosophy I criticized in Chapter One.

In this chapter, I will attempt to *bridge* the explanatory gap. I will attempt to do this in two ways. First, I will bridge the gap by using the general theoretical schema I advanced in Chapter Two to frame a type identity theory of pain. In that chapter, I showed how qualitative features of the experience of pain (e.g., pain submodality) can be naturally type identified with physiological features of the NEIM. These type pain identities are supported by *general* theories of the nervous, endocrine and immune systems, in concert with *general* theories of allostasis, homeostasis, stress and wounds, all of which, in turn, are constrained by the overarching theory that biological individuals are complex adaptive systems. I argued that this schema challenges the supposed *metaphysical gap*. My next attempt to bridge the gap is the claim that the supposed gap assumes that type identity theory is required to reconstruct type pain identities as formal inferences from natural laws. Based on actual scientific practice and philosophical considerations concerning explanatory levels, I will show that this is a false assumption.

My novel approach to bridging the explanatory gap is as follows. First, I will not attempt to bridge the gap by type identifying mechanism with pain *qualia*. Instead, I intend to bridge the gap by invoking the assumption that pain is type identical with specific NEIM mechanisms that are the *cause* of *pain facial expressions*. Recall that type identity theory of

mind is *realist* about pain qualia. Pain is causally responsible for behaviours such as facial pain grimaces and screaming, and for pain verbal self-report, which is also *pain behaviour* (Williams & Craig, 2006). Pain type identity theory does not exclude pain or make it disappear: pain qualia are *real*. And so are pain behaviours.

Philosophically, this assumption is supported by the Causal Closure of the Physical (Papineau, 2009). For, if causal closure were not true, then some physical effects (e.g., pain facial expression) would not be determined by prior physical causes (e.g., pain), but by ontologically unique mental causes such as nonphysical qualia. Thus, to deny this assumption and the Causal Closure of the Physical implies endorsing the unattractive philosophy that pain is epiphenomenal, having no causal powers, but appearing to have them because of its association with mechanism (Jackson, 1981, 1985). So, in type identifying pain with mechanism that is the assumed cause of pain facial expression, I take it to be the *best theoretical explanation* of that assumption.

Second, to bridge the explanatory gap, it needs to be located. The gap is not, however, *between* pain facial expression and mechanism spanning *different* explanatory levels. For example, Sullivan (2001) thinks that because pain is not in mind or body, it is therefore *between* minds and bodies as a special form of social behaviour. So, in addition to a category consisting of mental states and a category consisting of bodily states, there appears to be, according to Sullivan (2001), an ontologically *unique* category consisting of social pain behaviours. Views of this kind violate Occam's razor, and the theoretical value of simplicity, and should not be affirmed. In contrast, type identity theory of mind asserts a category consisting of mental states and a category consisting of physiological operations, and claims that one of the categories is reducible to the other. In the case of pain facial expression, the category consisting of facial behaviours is reducible to the category of physiological operations. A reduction of this kind is informative if what are identified in a statement of type

identity are two *delineations* of the same thing or operation (e.g., Craver & Bechtel, 2007; Feigl, 1958; Place, 1956; Smart, 1959). Thus, the location of the explanatory gap is not between the pain facial behaviours and the mechanism. Instead, it comes to light when the *identity conditions* of pain facial behaviours *and* responsible mechanisms are best explained from available descriptions (Craver & Bechtel, 2007; Sidelle, 2008; Wetzel, 2008). These delineations, which are mostly based on neuroscientific and psychophysical correlations, are type identified and then arranged together against the background of the general descriptive metaphysic I advanced in the previous chapter. Once such delineations are available, it will be evident that the delineated behavioural and physiological mechanisms have exactly the same identity conditions; that they are type identical.

2 Bridging the Explanatory Gap: a Polyvagal-Type Identity Theory of Pain Facial Expression

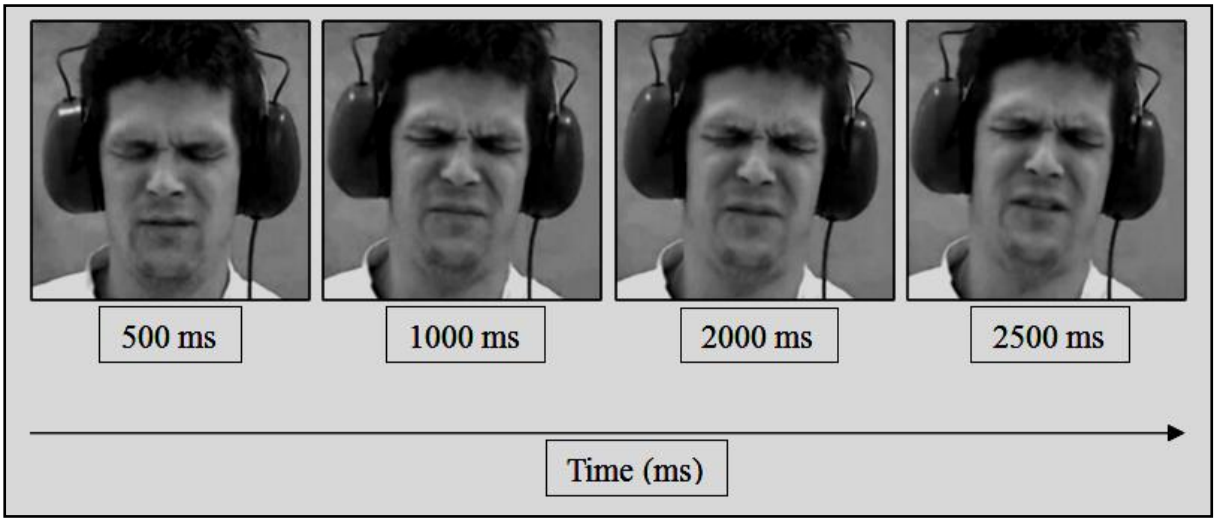
2.1 Overview of a Polyvagal-Type Identity Theory of Pain Facial Expression

In Chapter Two, I characterized pains as individuated according to somatosensory, emotional and cognitive qualia. I claimed that pain qualia are type identical to the operations of the NEIM. The experience of pain is causally responsible for a variety of type behaviours ranging from diverse forms of nonverbal actions to the use of language. Nonverbal type behaviours include vocalizations (screaming, moaning, and crying); nonverbal qualities of speech (amplitude, intonation); visible bodily actions (muscle tension, involuntary reflexes, intentional action); and facial expression (Craig et al. 2011; Keefe et al. 2001). The facial expression of pain is a complex multidimensional type of behaviour that involves somatosensory, negative emotional and cognitive features (e.g., Craig et al. 2011; Kunz et al. 2011a; Kunz et al. 2011b; Williams, 2002). The type features of pain I will mechanically

decompose concerning pain facial expression are: somatosensory (i.e., submodality, intensity, timing and duration); negative emotional (i.e., unpleasantness); and cognitive (i.e., modulation).

A well-established scientific literature exists on coding facial expression to quantify pain using the facial action coding system (FACS) (Ekman & Freisen, 1978). This literature reveals the specific and prototypical facial pattern of pain involves the corrugator (lowers brow), orbicularis oculi (tightens eyelids, raises cheeks), levator (wrinkles nose, raises upper lip) and zygomaticus (pulls up lip corners) facial muscles (Figure 6).

Figure 6: The Specific and Prototypical Facial Expression of Pain from Onset of Noxious Input, Across Time, in Milliseconds (ms), Adapted From Lamm et al. (2008)



The assumption that pain is type identical with specific NEIM mechanisms that are the cause of pain facial expressions is supported by many psychophysical and behavioural studies on pain facial expression in humans and nonhuman mammals (Craig et al. 2011; Prkachin, 2009, for review). The pain face is reliably observed as the response to acute clinical and experimental pain, including laboratory induced pain (e.g., Craig & Patrick, 1985), venipuncture (e.g., Grunau & Craig, 1987), and injections (e.g., Lilley et al. 1997), in

addition to exacerbations of chronic pain, including chronic lower back pain (e.g., Hadjistavropoulos & Craig, 1994), muscle contraction headaches (e.g., Iezzi et al. 2000), surgical repair (e.g., Hadjistavropoulos et al. 2002), and shoulder pathology (e.g. Prkachin & Mercer, 1989). It has been observed in a broad range of special populations, including neonates (e.g., Grunau et al. 1987), individuals with intellectual disabilities (Hadden et al. 2002), normal older adults (Kunz et al. 2007), individuals with dementia (Kunz et al. 2008), individuals with autism (Nader et al. 2004), individuals with temporomandibular disorder (LeResche et al. 1992), and in nonhuman mammals (Keating et al. 2012; Langford et al. 2010; Leach et al. 2012; Sotocinal et al. 2011). The pain facial expression can be type differentiated by untrained observers from facial responses to unpleasant, but non-noxious events such as disgust (e.g., Simon et al. 2008). Facial displays of pain rapidly diminish when analgesics are applied (e.g., Taddio et al. 1997). The rapidity of pain facial display onset and offset is partly explained by the function of the myelinated vagus nerve, as characterized in Polyvagal Theory (Porges, 2001, 2006) (see section 3.3 below).

Polyvagal Theory type identifies mammalian behaviour with mechanism (Porges, 2001, 2006), and therefore offers a suitable general background theory for typing pain facial expression with the operations of the NEIM. According to polyvagal theory, evolution of neural control within the autonomic nervous system (ANS) has tracked three stages, each revealing a specific behaviour. In the first stage, the ancient unmyelinated visceral vagus nerve that enables digestion could respond to danger only by reducing metabolic output and producing immobilization behaviours. In the second stage, the sympathetic nervous system (SNS) made it possible to increase metabolic activity and inhibit the visceral vagus nerve, thus allowing fight/flight behaviours following perceived threat. The third stage, which is *uniquely* mammalian, involves a *myelinated* vagus that can rapidly control cardiac output to enable spontaneous interaction (i.e., rapid engagement or disengagement) with the

environment. The mammalian vagus nerve is neuroanatomically connected to the cranial nerves that control environmental engagement through facial expression and vocalization and also cranial nerves V and VII originating in medullary nucleus ambiguus (NA).

Medullary NA is the source of cell bodies for somatomotor pathways travelling through several cranial nerves including the glossopharyngeal (IX) (control of pharyngeal muscles), vagus (X) (control of the muscles of the pharynx and larynx), and accessory nerves (XI) (control of the neck muscles). These cranial nerves evolved from primitive gill arches. In addition to these somatomotor functions, these branchiomeric (i.e., evolved from primitive gill arches) cranial nerves also control the visceromotor allostatic and homeostatic functions type identified with salivation, tearing, breathing, and heart rate. With evolution, the interaction of the ANS with the HPA, nervous, endocrine and immune systems change to maximize response to stressors, including wounding and nociception (Chapman et al. 2008; Porges, 2001, 2006). Based on this general theoretical framework, pain facial expression is a type of autonomic response caused by a type of sensory signaling (nociception). That is, it is an evolved component part of the phylogenetically recent *brain-heart-face mechanism* (Porges, 2001, 2006).

3 A Mechanistic Explanation of Pain Facial Expression

Given this polyvagal framework of pain facial expression, I will now describe the neurophysiological mechanisms that I believe are currently best type identified with pain facial expression. This task involves providing coarse-grained accurate descriptions of how the pain face can be type explained by specific operations of the brain-heart-face mechanism, as characterized in polyvagal theory (Porges, 201, 2006). Following my approach in Chapter Two, I will delineate below coarse somatosensory (i.e., submodality, intensity, timing and duration), negative emotion (i.e., unpleasantness) and cognitive (i.e., modulation) features as

embodied in the type relationships between pain facial expression and the brain-heart-face mechanism.

3.1 Pain Submodality and Emotion

Pain facial expression is uniform across experimental pain submodalities including cold, electric shock, pressure, and ischaemic pain (Craig et al. 2011; Prkachin, 1992, 2009; Williams, 2002). Prkachin (1992) observed that lowered brow, tightened eyelids, raised cheeks, wrinkled nose, raised upper lip and pulled up lip corner actions were present across all tested pain stimuli (see Figure 6 for prototypical pain face across time). Other reliably observed pain facial actions across pain submodalities are oblique lip pull, jaw drop, mouth opening, and horizontal mouth stretch (Botvinick et al. 2005; LeResche & Dworkin, 1988; LeResche et al. 1992; Prkachin, 1992). Acute exacerbation of chronic musculoskeletal pain shows a reliable pain face expression (Craig et al. 1991; LeResche & Dworkin, 1988; Prkachin & Mercer, 1989; Prkachin et al. 1994). Moderate correlations have been reported between facial expression measures and self-reported chronic disability (Prkachin & Mercer, 1989), and verbal self-report chronic pain assessments (Craig et al. 1991). Currently, it is not known whether these relationships exist in other chronic pain subtypes, such as fibromyalgia or neuropathic pain (Williams, 2002).

In a cognitive behavioural study, Kunz et al. (2011b) used a reliable psychological strategy (suggestions) to differentially alter the somatosensory and emotional features of pain. The authors found that suggestions for either increased pain unpleasantness or pain sensation evoked specific changes in facial expression, irrespective of pain submodality. Facial actions around the eyes were closely correlated with pain somatosensation, whereas facial actions of the nose, upper lip and also eyebrows were closely correlated with the unpleasantness or aversiveness of pain. Pain facial actions are under tight neural control through the

somatomotor component of the brain-heart-face mechanism: upper face innervation is bilateral and arises from the supplementary motor area (M2) and the rostral cingulate motor area (M3). Lower face innervation is contralateral and arises from primary motor cortex (M1), ventral lateral premotor cortex, and the caudal cingulate motor cortex (M4) (Morecraft et al. 2004). Thus, human pain facial movements of the nose, eyebrows and upper lip are type identical with negative emotional aspects of pain and activation of M1, M2, M3, whereas facial movements around the eyes are type identical with somatosensory aspects of pain, and activation of M2 and M3. Evolution of cranial anatomy enabled a highly integrated facial representation of the multidimensional experience of pain. These identity claims are heuristic to emphasize that they are *assumptions* that then guide subsequent empirical investigation (McCauley & Bechtel, 2001).

The unique integration of pain in the mammalian face appears paralleled within cortical targets in the paleospinothalamic tract. In an fMRI study, Kunz et al. (2011a) found strong activation in M1, S1, AINS, and ACC in the within-subject analyses comparing trials with and without facial expression in facial expressive subjects. Differences in verbal pain self-report did not explain the variability in pain facial expressions and the corresponding cortical activation because verbal pain self-report was not associated with pain facial expressiveness and did not differ between painful trials with and without facial expressions. Based on the type identity I claimed between pain unpleasantness and specific parts of the paleospinothalamic tract, the results of Kunz et al. (2011a) suggest that facial expression uniquely represents pain emotion compared to verbal pain self-report (Craig et al. 2011; Prkachin, 2009; Williams, 2002). Electrocortical evidence for preferential processing of dynamic pain expressions compared to other emotional expressions adds further support to this hypothesis (González-Roldan et al. 2011; Reicherts et al. 2012).

Pain through dynamic facial expression is characterized by mobilization. Thus, pain facial expression is type identified with the defensive fight-flight behaviours the NEIM that functionally increases metabolic output by increasing sympathetic, HPA and immune excitation. Simultaneous with these changes is a withdrawal of the myelinated vagal pathways in the brain-heart-face mechanism that is the vagal brake. Pain is always personal, but it is also social: it invariably is expressed in complex social environments, and manifest pain that leads to caregiving requires an ability to restrain facial and bodily mobilizations (Craig et al. 2011; Hadjistavropoulos et al. 2011; Williams, 2002; Wittgenstein, 2009). For example, during mammalian play, a playmate may sustain a wound. In full-contact sports, one player may be hit with an elbow or knee in the face. A fight may occur if the player who accidentally hits another in the face walks away without mitigating the pain through a *face-to-face* display of care and empathy. Similarly, when puppies play, one playmate may accidentally bite too hard and cause pain in the other. Play will likely stop if the puppies do not make face-to-face contact after this event. Face-to-face contact alerts the individuals involved that the intentionality of the behaviour is benign, and facial muscle activity influences the neural mechanisms that regulate visceral qualia (e.g., Gellhorn, 1964; Levenson et al. 1990). Thus, the mammalian face is necessary in understanding mobilization as play and not aggression (Porges, 2001, 2006; Porges & Furman, 2011). Although pain facial expression shares parts of the NEIM involved in fight-flight behaviours, expressed pain is social and requires dynamic neural regulation of state to expedite safe interactions and recovery. Hence, both sympathetic activation to increase metabolic output to enable pain behaviours and the vagal brake to restrain mobilization and to support caregiving are recruited to ultimately restore allostasis and homeostasis. Section 3.3 below links these ideas in relation to pain modulation.

The compromised brain-heart-face mechanism is correlated with a change in autonomic regulation type identified as a reduction in the influence of the myelinated vagus on the heart (Porges, 2001, 2006). This results in difficulties in behavioral state control with a loss of neural control to facial muscles regulating the comparatively flat negative emotional expressions often seen in clinical disorders (Porges, 2001, 2006; Porges & Furman, 2011). Since pain is part negative emotion, I would anticipate similar type identity relationships between autonomic state during pain and facial control in psychiatric populations, though this hypothesis has yet to be experimentally tested.

3.2 Pain Intensity

The intensity of pain is embodied in pain facial expression, both in the number of specific facial actions involved, and in the urgency or vigour of the expression (Craig et al. 2011; Prkachin, 2009; Williams, 2002). For example, Lints-Martindale et al. (2007) and Reicherts et al. (2013) found that facial responses linearly increase in intensity and frequency in relation to the intensity of the noxious stimulus. This additive relationship between intense pain qualia and pain facial displays has also been reported in nonhuman mammals (e.g., Keating et al. 2012; Langford et al. 2010; Sotocinal et al. 2011), and in congenitally blind and sighted individuals (Kunz et al. 2012). However, Kunz et al. (2012) also found that blind individuals were less capable than sighted individuals of deliberately modifying pain intensities in their facial expressions. While severe pain intensity may be evolutionarily prepared in pain facial expression, conscious modulation of pain intensity in the face may also require experiential visual learning (Kunz et al. 2012). Lower pain intensities are shown in narrowing of the eyes and brow lowering, with greater pain intensities linearly recruiting more vigorous displays in these actions and in the lower face (Craig et al. 2011; Prkachin, 2009; Williams, 2002). Some individuals facially respond to lower intensity levels of painful

stimulation than others (Prkachin & Craig, 1985). For example, in a psychophysical experiment, Prkachin (1992) found that the intensity of orbital tightening varied with pain intensity across all tested noxious stimuli. In pain observation settings, pain faces are judged as more unpleasant than anger or neutral faces across high, mild and low intensity levels (González-Roldan et al. 2011); higher reported pain intensity contributes to decreased visual attention to pain faces versus neutral faces (Vervoort et al. 2013).

The AINS has been reliably shown to be involved in communicating the diffuse feedback from the viscera into conscious awareness and facial behaviour. According to Critchley (2005) and Porges (2001, 2006), visceral and autonomic states are represented in the AINS and contribute to aversive noxious and non-noxious somatosensory and negative emotional qualia, such as the personal experience and observation of disgust (Wicker et al. 2003). Many human and nonhuman studies show that activity in the AINS is robustly correlated with autonomic and visceral information (e.g., Mesulam & Mufson, 1982; Mesulam, & Mufson, 2004; Saper, 1982, 2002).

In the personal experience of pain, AINS discharge increases linearly to the intensity of the painful stimulus (e.g., Peyron et al. 2000), to subject verbal self-reports of pain intensity (e.g., Coghill et al. 1999), and to increased pain facial vigour (Kunz et al. 2012). In a relevant fMRI study, Saarela et al. (2007) imaged brain activity in subjects as they observed static photographs of faces of pain patients. The represented pain in the visual stimuli was temporarily intensified for a few seconds. Saarela et al. (2007) found the novel result that activation strength in the observer's AINS was strongly correlated with the intensity estimates for pain in patient facial expression. Put simply and economically, pain intensity is embodied in pain facial expression; at the level of the mammalian brain, in both observers and individuals in pain, pain intensity in facial expression appears to be type identical to the operations of the AINS, an important part in the brain-heart-face mechanism.

3.3 Pain Timing and Duration

In clinical and experimental settings, the pain face is observed to rapidly appear following noxious stimulation, and diminish concurrent with cessation of the noxious stimulus following administration of analgesics (e.g., Craig & Patrick, 1985; LeResche & Dworkin, 1984; LeResche et al. 1992; Taddio et al. 1997). The brain-heart-face mechanism is an integrated system with both a somatomotor part controlling the striated facial muscles and a visceromotor part controlling the heart through a myelinated vagus nerve (Porges, 2001, 2006). When the vagal tone to the cardiac pacemaker is high, the myelinated vagus acts as a *brake* or restraint limiting heart rate. Rapid inhibition and disinhibition of vagal tone to the heart supports the rapid mobilization of facial muscles and formation of the pain face concurrent with pain onset (Porges, 2001, 2006). In humans and nonhuman mammals, the main vagal inhibitory pathways in the myelinated vagus originate in the NA. The vagal brake supports the low-metabolic requirements involved in the rapidly appearing and disappearing pain face. Withdrawal of the vagal brake is strongly correlated with the rapid appearance of the pain face; reinstatement of the vagal brake is strongly correlated with the rapid diminishment of the pain face. These correlations are not unique to pain facial expression; similar relationships hold with regard to the vagal brake and the timing and duration of aversive, but non-noxious emotional facial expressions (e.g., Pu et al. 2010), and positive emotional facial expressions (e.g., Kok & Fredrickson, 2010).

In terms of the function of rapid pain face onset and offset, the vagal brake makes it possible for the individual in pain to quickly disengage from source of wounding and pain, concurrent with the rapid appearance or diminishment of pain facial expression, which may offer temporary access to additional metabolic resources to aid allostasis, recovery and self-soothing behaviours, with likely involvement from care givers. Concerning aid from others,

the vagal brake reliably maps onto specific interaction types observed in mammalian pain events. In pain events comprising the individual in pain and care givers, mammalian behaviour is typed according to interpersonal communication through facial expressions, vocalizations, head and hand gestures (Hadjistavropoulos et al. 2011; Porges, 2001, 2006; Williams, 2002). A relevant feature is the rapid 'switching' of temporary engagement to temporary disengagement behaviours between the individual in pain and care givers. This interaction type may involve care givers speaking to the one in pain, and then quickly switching to listening; for the one in pain, looking into the face of the care giver, and then quickly switching to vocalizing (Craig et al. 2011; Hadjistavropoulos et al. 2011; Porges, 2001, 2006; Williams, 2002). The brain-heart-face mechanism allows the one in pain and the care giver to *get the timing right.* Some philosophers and neuroscientists claim that evolutionary neurobehavioural solutions to timing problems such as these are implicated in the origin of empathy and ultimately consciousness itself (Churchland, 2002, 2011; Cole, 1998; Decety, 2011; Engen & Singer, 2012; van Rysewyk, 2011).

However, if pain is severe or chronic and the vagal brake is withdrawn (or dysfunctional), the concurrency of increased pain facial expression, cardiac output, and other mobilization behaviours (i.e., increased SNS and HPA output), means that, if care giving is to succeed in promoting healing and recovery, the *caregiver's* vagal brake must be dynamically reinstated (Chapman et al. 2008; Hadjistavropoulos et al. 2011; Porges, 2001, 2006). By applying their own vagal brake, care givers may regulate their own visceral distress and thereby succeed in allocating valuable metabolic resources to communicate safety to the one in pain (and themselves) through calming facial and head behaviours, eye gaze, and prosodic vocalizations (i.e., increasing the vagal brake decreases SNS and HPA output). Since the vagal brake of the person in pain has been provisionally withdrawn, the care giver is effectively an integrated *external* brain-heart-face mechanism (cf. Tantam, 2009, the

‘interbrain’). Thus, the muscles of the pain face are best explained here as neural timekeepers detecting and expressing features of safety and danger that cue the one in pain to quickly disengage from source of wounding and pain, simultaneous with the rapid appearance or attenuation of pain facial activity, and also cue others who can facilitate allostasis. The clearest and most prudent explanation of these delineations is that pain facial expression is type identical to the operations of the brain-heart-face mechanism.

3.4 Pain Modulation

Since it appears explanatorily best to type identify pain facial expression with the operations of the brain-heart-face mechanism, its spontaneous display following noxious stimulation would therefore almost always be entirely unconscious and involuntary (Craig et al. 2011; Porges, 2001, 2006; Prkachin, 2009; Williams, 2002). This type identity claim is supported by developmental data showing type identity relationships between the brain-heart-face mechanism and facial expression of pain and emotion in neonates, babies and infants (Craig et al. 2011; Grunau & Craig, 1987; Porges, 2006; Porges & Furman, 2011; Williams, 2002), in individuals with severe cognitive disabilities (Hadden et al. 2002), individuals with compromised language ability (Kunz et al. 2008; Nader et al. 2004), and from comparative data in nonhuman mammals (Keating et al. 2012; Langford et al. 2010; Leach et al. 2012; Sotocinal et al. 2011).

Kunz et al. (2012) investigated involuntary (i.e., unconscious) and voluntary (i.e., conscious) pain facial expressions in congenitally blind and sighted individuals to assess the contributions of evolution (Williams, 2002) or visual learning to facial display (Ekman & Friesen, 1971, 1978). The authors found that the repertoire of unconscious pain facial expressions was comparable in blind and sighted individuals; however, blind individuals were less capable of consciously presenting their ‘optimal’ pain face according to

experimental instructions. Thus, although the conscious control of pain facial expression appears to rely on visual experiential learning, the repertoire of facial muscles being used during pain is evolutionarily prepared (Kunz et al. 2012).

The extent to which the face is *actually* used to display an individual's pain or emotion can vary, at least in human older children and adults (Craig et al. 2011; Porges, 2001, 2006; Porges & Furman, 2011; Williams, 2002). The absence of facial expression may contradict verbal self-reports indicating strong pain (Hill & Craig, 2002) or strong negative emotional, but non-noxious experiences (e.g., Gross & Levenson, 1993, 1997). Numerous studies show audience effects on pain facial expression in adults (e.g., Badali, 2000, 2008) and infants (e.g., Din et al. 2009; Horton et al. 2010). Adults who are alone rather than in the company of adult strangers are more inclined to show the full pain face. Pain facial expressiveness varies from high expressiveness to stoicism (Craig et al. 2011; Prkachin, 2009; Williams, 2002). In a fMRI study, Kunz et al. (2011a) found that pain facial expression was inversely related to frontostriatal activity, which is consistent with the hypothesis that this cortical region partly controls the suppression of facial displays (e.g., Morecraft et al. 2004).

According to polyvagal theory, the relationship between visceral state and modulation of facial expression reduces to the brain-heart-face mechanism, since the metabolic costs necessary to modulate the facial muscles (e.g., suppression or amplification of pain facial expression) during emotional or pain states mechanically decomposes to supporting changes in autonomic state. Gellhorn (1964) and Levenson et al. (1990) found that facial muscle activity influences the mechanisms in corticolimbic pathways that control visceral qualia. This phenomenon is commonly seen in human and nonhuman mammals in threatening but pain-free settings including very young infants who use sucking behaviours to sedate, to older human mammals that use smiling, listening, conversation and ingesting to calm. In contrast,

walking away, or turning the head away from a threatening or dangerous event (e.g., a pain event) can cause a violent response (Porges, 2001, 2006; Porges & Furman, 2011).

Concerning psychophysical investigations of the relationship between pain, modulation of facial expression and autonomic state, in one psychophysical study, Lanzetta et al. (1970) instructed subjects to facially pose anticipating and receiving one of two levels of painful electric shock: either no shock, or "extremely intense, almost unbearable shock". In the study, a colour slide indicated to the subject both the level of shock and the facial posing condition on a given trial. For example, a red 1 signalled that a low shock was to be given, but that an *intense* pain should be facially posed, a green 2 signalled a moderate shock and a facial pose of no pain. The authors found that when the subjects facially posed intense pain, they judged the shocks as more intense (r_m = .769) and had larger skin conductance responses (a standard measure of autonomic state) both to the signals and to the painful shocks themselves (r_m = .716 and r_m =.866, respectively). Lanzetta et al. (1970) concluded that the results count as evidence of a necessary relationship between modulation of pain facial expression and consequent autonomic visceral state. In a similar psychophysical study, Kleck et al. (1976) found a decrease in skin conductance responding (r_m = .462, Study 1; r_m =.429, Study 2) and self-reported pain (r_m = .506, Study 1; r_m =.726, Study 2) when the attenuation of pain facial expression was caused by social cues; namely, the subject was told that he or she will be observed during painful stimulation. More research is needed in order to evaluate these findings in relation to type identity theory of mind.

4 Summary of Section 3

I hope to have made clear at a coarse level of explanation how somatosensory, negative emotional and cognitive features are type embodied in the relationship between pain facial expression and the brain-heart-face mechanism, as described in polyvagal theory

(Porges, 2001, 2006). Ultimately, the descriptive metaphysic offered in Chapter Two best accounts for these type pain identities. The type identification of pain facial expression with the brain-heart-face mechanism is nonetheless guarded because the mechanistic explanations and fundamental descriptive theories are still relatively immature. Even though evidence has not yet type identified all features of pain at a suitably *fine-grain*, I suggest that this is a trivial fact about the current status of scientific theories rather than a *special* fact about the relation between pain qualia and pain facial expression. The explanatory gap is *real.* But, I hope to have demonstrated its existence only reveals a pragmatic limit on our present explanatory accounts of pain facial expression and pain generally. Thus, we would be explanatorily justified in claiming that pain masochism is type identical to the NEIM, even if we did not yet know what the specific physiology of the mechanism is. We would also be permitted to assert that the qualia of pain masochism are entirely explained by – nothing more than – neurophysiological mechanism. Thus, concerning the type pain identity claims made in Chapter Two and in the current chapter, I believe the most coherent and parsimonious explanation of these stated relationships is that pain is type identical to mechanism.

5 Bridging the Explanatory Gap: Discovery of Type Identities, Mechanism and Levels

I will now argue that consideration of the nature of type identity claims and mechanistic explanation reveals that the problem of the explanatory gap is based on a false assumption. Recall that supporters of the gap puzzle assert that even if type identity theory of mind is true, it still needs to explain why and how a specific pain is type identical to a specific mechanism, rather than a different mechanism, or conversely, how and why a specific mechanism is not type identical with an entirely different type of experience (Levine,

1983, 2001). However, many philosophers believe type identity theory cannot offer such explanations because it is false. I criticized this assertion in Chapter Two. In this section, I will address the 'X = Y, but why not Z?' objection to type identity theory of mind.

Philosopher Levine (1983, 2001) asserts that a type identity claim must explain *why* a specific pain is type identical to a specific mechanism, rather than a different mechanism. This assertion is intended to include type identity claims describing conscious mental states, in line with the philosophical strategy of the explanatory gap; however, it can be further generalized to include *all* type identity claims, irrespective of physical domain. Using examples from other subfields of science, why is visible light electromagnetic radiation as opposed to something else completely? Why is planetary wind actually the outgassing into space of light chemical elements from a planet's atmosphere? Why is a flower a reproductive organ? At its most general, why is a thing the thing it is? Levine (1983, 2001) does not specify what type of explanations type identity theory is expected to provide for claimed identities, and what form of relationship is required between identities and more fundamental explanations. So, I will interpret the line of reasoning above to imply the view that, in order to bridge the explanatory gap, type identity theory of mind is required to reconstruct true type pain identities as *logical derivations* from assertions of natural *laws*. Thus, the expected explanation has the form of a sound deductive argument in which a type pain identity *follows* as a conclusion from premises which contain a law or laws of nature. This law must be a necessary premise in the inference because the inference of the type pain identity would not be valid if this premise were excluded. This interpretation of the explanatory gap closely resembles, but is not identical to, the *deductive-nomological model* (DN) of scientific explanation (Hempel, 1948, 1965; Nagel, 1949, 1961).

What is a law of nature? Based on DN, there are true generalizations that are only *contingently true* and those that are *laws*. For example, the generalization "All current

citizens of Australia are in pain" is, if it is true at all, only contingently true. By contrast, "All gases expand when heated under constant pressure" is a law. According to the DN model, the latter generalization can be used, together with information that a *specific* gas has been heated under constant pressure, to explain why it has expanded. By contrast, the former generalization concurrent with the information that a specific person is an Australian citizen cannot be used to explain *why* that person is in pain. Accordingly, the challenge type identity theory must meet is to specify a natural law or laws which explain that pain is the allostatic stress response of the NEIM, rather than a different mechanism. If that challenge can be met, the explanatory gap can be bridged – in principle.

5.1 Explaining Pain: Natural Mechanisms or Natural Laws?

However, this view of the explanatory gap, if it indeed accurately reflects Levine's (1983, 2001) position, faces two problems. One problem is that it is based on a false assumption; that is, the assumption that type pain identities logically follow from explanations – is false. In practice, neuroscience (and science generally) does *not* offer explanations for basic type identities (Baars, 2012; Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993; Churchland, 1989, 2002). Consulting the pain science literature, it is evident that the term scientists most commonly use in explanatory delineations is *mechanism.* In that vast literature spanning several centuries, there are many hypothesized mechanisms to explain pain, including nociceptive, inflammatory, and pathological subtypes of pain, and *shared* mechanisms across pain subtypes, as well as mechanisms to explain pain-related phenomena such as pain empathy or pain synesthesia. More broadly, I conducted a Google

Scholar search of peer-reviewed journal articles using the terms *law* and *pain* between 2000 and 2012 and only found a *single* research project comprising 3 articles, e.g., [14]:

1. Omoigui, S. (2007).The biochemical origin of pain – Proposing a new law of pain: the origin of all pain is inflammation and the inflammatory response. Part 1 of 3 – A unifying law of pain. *Medical Hypotheses, 69*(1), 70-82.

By contrast, I conducted a separate search on Google Scholar of journal articles using the terms *mechanism* and *pain* between 2000 and 2012 and found 1,280,000 such articles. Of this total, 466 of those articles used both mechanism and pain in the title. Here is a sampling of five titles (2-6) that offered mechanistic explanations of pain:

2. Mercadante, S., Fulfaro, F., & Casuccio, A. (2002). Pain mechanisms involved and outcome in advanced cancer patients with possible indications for celiac plexus block and superior hypogastric plexus block. *Tumori, 83*(3), 243-245.
3. Coull, J. A. M., Boudreau, D., Bachand, K., Prescott, S. A., Nault, F., Sík, A., Koninck, P. D., & Koninck, Y. D. (2003). Trans-synaptic shift in anion gradient in spinal lamina I neurons as a mechanism of neuropathic pain. *Nature, 424*, 938-942.
4. Wadachi, R. A., & Hargreaves, K. M. (2006). Trigeminal Nociceptors Express TLR-4 and CD14: a Mechanism for Pain due to Infection. *Journal of Dental Research, 85*(1), 49-53.
5. Schmidt, B. L., Hamamoto, D. T., Simone, D. A., & Wilcox, G. L. (2010). Mechanism of Cancer Pain. *Molecular Interventions, 10*(3), 164-178.

[14] This search was conducted on Google Scholar, December 28, 2012.

6. Zeilig, G., Enosh, S., Rubin-Asher, D., Lehr, B., & Defrin, R. (2012). The nature and course of sensory changes following spinal cord injury: predictive states and implications on the mechanism of central pain. *Brain, 135*(2), 418-430.

Like explanations of biological phenomenon offered in the life sciences generally, mechanistic explanations of pain in peer-reviewed science journal and book publications depart in three main ways from DN or nomological explanations (Bechtel & Abrahamsen, 2005; Bechtel & Richardson, 1993; Craver, 2007; Darden, 2006; Glennan, 2002; Machamer et al. 2000; Sarkar, 1998; Schaffner, 1993; Woodward, 2003). One, mechanistic explanations offered in pain science are not limited to logical derivations and linguistic representations. Pain scientists and theorists always use diagrams and figures to delineate pain mechanisms and systems, as I have done in the current project. This suggests that mechanistic explanation possesses greater epistemic resources compared to DN or other nomological theories of scientific explanation. Two, explanations or models of pain mechanisms are advanced for specific pain qualia (e.g., pain intensity, pain duration), or pain subtypes (e.g., nociceptive pain, inflammatory pain, pain disorders), and are not represented in terms of universally quantified assertions. Instead, generalization involves investigating both the similarities of new pain explanations to those already available, and the differences between them, typically across human and multiple nonhuman animal species. Three, since biological mechanisms are comprised of component parts and operations implies that both discovering and testing of mechanistic explanations involves – unlike nomological approaches – replicable experimental procedures for the decomposition, localization, and recomposition of pain mechanisms (described in Chapter Two, section 2).

As I have characterized it, the assumption of the explanatory gap puzzle views explanation of pain as a logical relation that is in principle possible although it is not

happening in practice. If explanation in this sense is not happening in pain science and in biology generally, then significant doubts arise as to whether it best explains pain biological knowledge and practice. Thus, the assumption that type pain identities logically follow from laws appears false because it relies exclusively on formal, rather than substantive issues. In the following section, I will address the second problem facing Levine's (1983, 2001) formulation of the explanatory gap, a problem confounding the relationship between type identity claims, mechanism and explanatory levels.

5.2 Discovering Type Pain Identities, Levels and Mechanism

One way of stating the explanatory gap problem is: can we explain *how* to make *mental* pain out of *bodily* physiological mechanisms? According to this formulation of the puzzle, the existence of an explanatory gap shows our incomplete understanding of how pain consciousness might rely upon a nonconscious neurophysiological substrate. This way of stating the gap appears to represent pain and mechanism, or most broadly, mind and body, as occupying different *levels*. Can they be bridged? If the interpretation of the gap in terms of levels is correct, the experience of pain and neurophysiological operations cannot ever be type identical. In this section, I will argue that this implication is mistaken, and that a correct understanding requires consideration of the relationship between type identity claims, mechanism and levels. So, what is a level?

The approach I adopted in this project is to consider mechanisms directly in explaining the nature of pain, as introduced in Chapter Two, section 2. Mechanistic explanation treats as *one* level the mechanism and the other parts (mostly other mechanisms) with which it communicates and as a different, *lower level* those component parts and operations that are involved in the functioning of the mechanism (e.g., Bechtel &

Abrahamsen, 2005; Craver, 2007; Darden, 2006; Glennan, 1996, 2002; Machamer et al. 2000; Woodward, 2003). Accordingly, scientific or philosophical analysis of a mechanism is an *interlevel* investigation: decomposing a mechanism into its component parts and operations is to move to a lower level, and thus is described as *reductionistic* (Bechtel & Abrahamsen, 2005; Craver, 2007; Darden, 2006; Woodward, 2003). However, the type identity claims which I am focusing on in this project are *within* a level. They relate two different descriptions as referring to either the same mechanism or the same component (i.e., part or operation) of a mechanism. Thus, a type identity claim asserts a distinctive relation between something and itself. For a type identity claim to be descriptive (i.e., informative), what are identified in an assertion of type identity must be two delineations of the same thing or operation. Since the same thing is not at two *different* levels, a type identity is not reductionistic (Craver, 2007; Hill & McLaughlin, 1998; Hooker, 1981; Papineau, 2002). The NEIM is not at a different level than pain anymore than the outgassing of light chemicals from a planet's atmosphere into space is at a different level than planetary wind. Thus, from the slant of type identity theory of mind, Levine's (1983, 2001) formulation of the explanatory gap as applied to pain; namely, "Why is pain what it is?" can now be given a reasoned reply: *pain is what it is because it simply is*. Science discovers type identities, but the type identities it finds simply are the way things are. Thus, the demand that type identity theorists explain why a specific pain is type identical to a specific mechanism, rather than a different mechanism, is mistaken if it entails that pain and mechanism are on different levels. Since this reasoning may appear to lead to a somewhat paradoxical conclusion, I offer the following comparison. If I discover that water is type identical to H_2O, I will explain why I believe this type identity by offering evidence. But if someone enquires, "Why is water type identical to H_2O?" no independent answer can be given; that is simply the way water is. Thus,

this question follows from the mistaken assumption that type identities are themselves reductionistic.

However, the enquirer may insist that invoking type identity does not dispose of the requirement for explanation (Levine, 1983, 2001). Even if two delineations *in fact* pick out one and the same thing or mechanism, we may still fairly anticipate some explanation of that type identity. In other instances of scientific type identities (e.g., visible light, temperature), there is an explanation that accounts for the type identity. Thus, it appears reasonable to anticipate the same concerning the claimed type identity of pain with the NEIM. In response to this objection, and returning to the type identity of water, there may be an explanation of why people erroneously believed that water was phlegm. However, there is no explanation of why water *is* water; of why water is type identical to H_2O. *It simply is.* Equally, assuming pain science discovers that a certain operation of the NEIM is type identical to the rare experience of pain synesthesia (Fitzgibbon et al. 2010) there will be no additional explanation of why that specific mechanism is type identical to pain synesthesia (Craver, 2007; Hill & McLaughlin, 1998; Papineau, 2002).

5.2.1 Decomposing Pain Mechanisms: The Essential Role of Neuroscience

Type identifying the experience of pain with the NEIM makes possible additional investigations into how the NEIM is responsible for pain and correlated pain qualia by further *decomposing* it. This same point corresponds to how the NEIM is responsible for pain that is the cause of specific types of behaviours, such as pain facial grimaces, and also subtypes of pain facial expressions, including involuntary and voluntarily controlled pain facial displays (Craig et al. 2011; Williams, 2002). When psychological resources reach their explanatory limits, further decomposition is conducted neuroscientifically (e.g., Bechtel & Abrahamsen, 2005; Craver, 2007). For example, roughly 20 years ago, psychological researchers had reliably delineated the structure and function of voluntary and involuntary pain facial expressions, but whether primary motor cortex (M1) is involved in only one or in both of the subtypes was unknown (e.g., Rinn, 1984). Using fMRI, Kunz et al. (2011) found robust activation in M1 during the display of involuntary and voluntarily controlled pain facial expression, thus confirming its involvement in both pain facial subtypes. Further specific decomposition would involve investigating correlations between pain facial expression, brain subregions, and *cellular* and *molecular* operations, such as the involvement of inhibitory interneurons in making gamma frequency oscillations in networks of pyramidal neurons (e.g., Buzsáki & Draguhn, 2004; Sejnowski & Paulsen, 2006; Traub et al. 1998). For example, pain neuroscientists will need to establish how neurons in specific subunits of M1 communicate with each other and neurons in sensorimotor pathways to generate oscillating networks that in turn produce lower-face movements during pain. Alternately, how neurons in subunits of rostral cingulate cortex (M3) make oscillating circuits that yield *upper*-pain face activity. While these questions have yet to be experimentally investigated, continued experimental and philosophical success in delineating pain and in relating its various aspects such as facial expression to neurophysiological operations suggests we are moving nearer and nearer to

bridging the explanatory gap. The capacity of type identities to enable further investigations means they are not necessarily conclusions of scientific research but can be hypothetical premises; that is, due to their potential to advance scientific research they can initiate scientific investigations (Eronen, 2013; McCauley & Bechtel, 2001).

5.2.2 Recomposing Pain Mechanisms: Interlevel Causation

Further to decomposing pain mechanisms, it is essential to *recompose* them in order to show how they operate in context. This involves unifying reductionistic pain research with pain research at higher explanatory levels (e.g., pain psychology, pain psychophysics, philosophy of pain). Some philosophers of science who endorse mechanistic explanation often appeal to *top-down causation* to understand how operations at high levels influence those at lower levels (e.g., Bechtel, 2011; Bechtel & Hamilton, 2007; Craver & Bechtel, 2007; Powell & Dupré, 2009). These philosophers have focused on describing variations between causal relations and interlevel relations. A regular feature of the world appears to be that in all instances of causation, the cause and the effect are independently placed in time so that the cause precedes its effect temporally by some action. According to Craver and Bechtel (2007), this apparent feature of causation does not hold of interlevel relations. If a component part of a mechanism is changed, the entire mechanism is changed, and a mechanism cannot be changed without some component part of it being changed. That is, the part and the mechanism alter concurrently: the two effects co-occur; they are not independent in time. Thus, when a causal variable alters a mechanism and thereby alters a component part, a host of other changes may occur in the mechanism, with the result that the mechanism is further changed and thereafter functions differently. For example, the three specific nonsense mutations S459X, I767X and W897X change the function of $Na_v1.7$ in the gene *SCN9A* with consequent complete loss of nociceptive input (i.e., CIP) (Cox et al. 2006; Cox et al. 2010).

According to Craver and Bechtel (2007), there is no 'top-down' or 'bottom-up' causation; there is only interlevel causation. The point they stress is the *level* at which the causation occurred, not the temporal sequence. If this view is correct, then both interlevel relations and type identity relations cannot be explained causally.

Thus, mechanistic explanation involves building understanding of levels (Bechtel, 2011; Bechtel & Hamilton, 2007; Craver & Bechtel, 2007; Powell & Dupré, 2009). Pain phenomena at the initial 'macro' level (e.g., pain facial expression) are decomposed into finer operations (e.g., upper-face expression, lower-face expression), which are subsequently type identified with operations within specific neural regions or structures (e.g., M3 control of orbicularis-oculi, M4 control of zygomaticus). Neurophysiological mechanisms at the molecular or cellular level, such as coordination of neural network oscillations, are concurrently delineated to explain their involvement in operations in specific contexts (e.g., M3 subunit activation of orbicularis-oculi in high empathy pain observers) (Lamm et al. 2008; Shackman, 2011). Type identity statements that successfully emerge from mechanistic pain explanation are therefore *between* different delineations of pain phenomena at the *same* explanatory level; that is, they are *intralevel*. And through the continued explanatory tasks of decomposition, localization and recomposition, pain scientists and theorists may gradually increase their understanding to levels such that delineations of experience and mechanism (or behaviour and mechanism), are seen to have exactly the same type identity conditions (Churchland, 2002; Craver & Bechtel, 2007; Sidelle, 2008). The success of such tasks implies that what might initially escape mechanistic explanation gradually contracts towards nothing (Sidelle, 2008). Thus, at the conclusion of successful pain research, the explanatory relation between the delineations on either side of the type identity sign is no longer puzzling: the explanatory gap has been bridged.

6 Conclusions

The explanatory gap puzzle correctly reveals our inadequate understanding of how pain might be explained in terms of the NEIM. Indeed, there *is* a gap in the naturalistic pain explanations currently on offer. However, the existence of the gap merely represents a pragmatic limit on our current explanatory successes. As described in Chapter One, some philosophers believe on *a priori* metaphysical grounds that pain cannot ever be explained in terms of neurophysiological mechanisms. Some of these philosophers further claim that pain is not explained by mechanism, as a matter of fact. However, since this metaphysical view was challenged in Chapters One and Two, the impossibility of bridging the gap as a matter of *principle* might be false. I attempted to bridge the explanatory gap in two ways: first, based on a novel polyvagal-type identity theory of pain facial expression situated in the broad theoretical framework developed in Chapter Two; second, I reasoned that supporters of the *in principle a priori* gap believe that type identity theory must build type pain identities as formal logical derivations from laws. I tried to show that this belief is false, based on actual scientific explanatory practices and philosophical considerations concerning explanatory levels. The explanatory relation between the descriptions on either side of the type identity sign is, at the end of successful pain research, no longer vexing: the explanatory gap has been bridged. In the next chapter of the project, I will consider a final challenge to type identity theory of pain. This is the radical view that successful pain research will show that pain is not *real*, and therefore ought to be *eliminated*.

4 Elimination of Pain?

Abstract

Type identity theory of mind supports philosophical intuitions about the causal powers of pain qualia such as the idea that pain is causally responsible for behaviours such as pain facial expression. Another philosophy of mind called eliminative materialism agrees with the identity theory that there is nothing more to pain than physiological mechanism, but denies that pain can be type identified with mechanism. This is because pain does not really exist. Thus, philosophical intuitions and everyday folk psychological explanations of pain are false and have no place in an accurate neuroscientific explanation of pain. In this chapter, I consider philosophical arguments for and against eliminative materialism of pain. If eliminativism is correct, then the version of type identity theory of pain I propose in this project is strictly false. While pain folk psychology has stimulated much scientific psychological research, which has led to improved clinical outcomes for some pain patients, our own intellectual history shows that any theory can appear successful or beneficial even when it is completely false. Moreover, neuroscience already demonstrates that many core folk pain claims are mistaken, while the truth of many others is not known. Eliminative materialism implies the unsettling consequence that the limit of what can be scientifically eliminated or reduced is much closer than we may intuitively believe. Intellectual history also offers modest encouragement for co-existence between a type identity theory of pain and folk pain psychology. Sometimes a mistaken theory may not be fully eliminated, and can survive in altered form in a new theory. Partial elimination and pain folk psychology can co-exist, albeit for a time. Thus, radical theory change is merely one end-point on a continuum among many theoretical possibilities. Still, theoretical accommodation cannot alter the realization

that all human knowledge is ultimately speculative and temporary. This insight fosters modesty about the truth of our current pain theories, both folk and scientific, and this pragmatic wisdom extends to the type identity theory of pain I offer in this project, while it encourages a humble optimism concerning our future theoretical prospects.

Keywords: chronic pain, clinical medicine, eliminative materialism, folk psychology, mirror neuron system, naturalism, pain, pain empathy, simulation theory, theory-theory, type identity theory of mind

1 Introduction

In Chapter One, I observed that type identity theory of mind supports *realism* about mental qualia as causative. A type identity theorist of pain views pain as causally responsible for such behaviours as facial grimaces and crying out, and also for pain verbal self-report. Accordingly, a pain type identity claim does not exclude pain or make it disappear – pain qualia are certainly *real*. The core point of the type identity theory of pain offered here is that every member of the type *pain qualia* is necessarily a member of the type *operations of the NEIM*; the two types are identical. The identity theory of mind is thought by some philosophers to best explain internal somatosensory states rather than those of belief, desire and anticipation whose individuation may rely on relations to actual things in the external world (e.g., Hill, 1991; Place, 1956; Smart, 1959; Wickforss 2007). However, in Chapter Two, I presented sufficient evidence to show that the somatotopy of S1 best explains the anticipation of pain in a specific body part. These 'patchy and fragmentary' mechanistic explanations (Schaffner, 2006) based on accumulating data can already inform pain identity claims and may ultimately explain everyday common-sense reasoning such as: "My leg has pain that is similar to what I experienced when my shoulder had pain, and because medical care relieved my leg pain, I anticipate it will relieve my shoulder pain". There appears no problem in principle here, only a significant challenge (Baars, 2012; Churchland, 1989, 2002; Demertzi & Laureys, 2012).

A different way of stating the relationship between pain and mechanism is to assert that there is *nothing more* to pain than physiological mechanism (i.e., the NEIM). That is, pain is 'nothing over and above' mechanism. This assertion amounts to a *minimal reductionism* about pain (Polger, 2011). Minimal reductionism concerning pain contrasts with dualist and emergentist philosophies according to which pains are dependent on neurophysiology but are nevertheless somehow 'more than' the neurophysiology on which

they depend (e.g., Chapman et al. 2002; Chapman et al. 2008; Nakamura & Chapman, 2002). Type identity theory of pain rejects emergentist philosophies of pain.

Another physicalist philosophy of mind which accepts minimal reductionism, like type identity theory, is *eliminative materialism* (EM). However, unlike type identity theory, EM *denies* that pain can be type identified with mechanism. This is because pain, as understood by the type identity theorist and everyday common-sense, *does not really exist*. Thus, philosophical intuitions and everyday common-sense claims about the subjectivity and causal powers of pain are false and ultimately have no place in a neuroscientific successor theory of pain (e.g., Churchland, 1981; Churchland, 1989; Dennett, 1978; Hardcastle, 1999; Stich, 1983). The relationships between type identity theory and EM concerning the core theses of type identity theory presented in this project are summarised in Table 3 (adapted from Polger, 2011).

Table 4: The Six Core Philosophical Theses of Type Identity Theory and Their Relation to Eliminative Materialism, Adapted From Polger (2011)

Philosophical Thesis	Type Identity Theory	Eliminative Materialism
Realism – Pain qualia are real.	Yes	No
Physicalism – Pain qualia are physiological.	Yes	Yes
Minimal Reductionism – Pain qualia are nothing more than physiological mechanism.	Yes	Yes
Type Identity – Pain qualia are type identical to physiological mechanism.	Yes	No
Naturalistic – Philosophies of pain are both metaphysical theories *and* scientific theories.	Yes	Yes
Theoretical – Metaphysical theories of pain can be assessed according to their theoretical virtues (e.g., simplicity), *and* competing empirical predictions.	Yes	Yes

The aim of this chapter is to critically assess the arguments for and against EM concerning pain. The first task of this chapter is to describe and assess three recognised arguments for EM. In section 3, I specifically consider philosopher Daniel Dennett's (1978) version of pain EM.

2 The Case for Eliminative Materialism

2.1 Theory-Theory and Folk Psychology

The central argument for EM begins with the idea that we use a *theoretical* framework to explain and predict human behaviour (Sellars, 1956). In philosophy of mind,

this idea is usually called the *theory-theory* (TT). TT views folk psychology (FP) as comprising specific theoretical claims and generalizations (and laws), described by our everyday common-sense psychological (i.e., mental) words such as 'belief', 'desire', 'recognition', 'fear', 'anticipate', 'memory' or 'pain'. FP generalizations are thought to describe the diverse causal regularities and relations of FP claims. FP pain generalizations have been documented in many studies of patient pain beliefs and cognitions (e.g., Butler et al. 2003; Eccleston et al. 1997; Goubert et al. 2011; McCrystal et al. 2011; Moseley, 2007; Williams & Thorn, 1989). They range from the simple to the complex:

1. Pain generally hurts and is unpleasant.
2. Pains generally are located in a part of the body.
3. People generally want pain to stop as soon as they feel it.
4. A change in the intensity of a pain is generally influenced by a change in the weather.
5. Wounding generally causes pain, and when the wound is completely healed, pain usually stops.
6. If a physician cannot determine a physical cause for a person's pain, then a physician will generally not believe that a person is really in pain.
7. If a person wants his or her pain to stop and believes that the best way to get the pain to stop is to consult a physician, then a person will generally consult a physician.

Aldrich and Eccleston (1999) asked 61 study participants to sort 80 statements describing 'the culture of pain'. Eight pain FP claims emerged from the Q factor-based analysis:

8. pain as abuse

9. pain as alien invasion
10. pain as coping and control
11. pain as homeostatic mechanism
12. pain as malfunction
13. pain as mental
14. pain and power
15. pain as self-growth
16. pain as spiritual growth

Aldrich and Eccleston (1999) derived common themes that cut across the eight claims. One shared theme was the idea that pain must offer *meaning*. Another set of themes consisted of how pain relates to self; specifically, whether or not pain can *change* the self.

TT claims that pain generalizations and claims like these operate in FP much like the generalizations and laws of *scientific* theories. However, the laws of FP are acquired more informally than scientific theories, as part of normal human development (e.g., Churchland, 1981; Hardcastle, 1999; Lewis, 1970, 1972; Roth, 2012; Stich, 1983). For example, children who observe their parents showing fear and behavioural avoidance to back-stressing tasks, such as lifting heavy objects, may adjust their understanding of that situation ("back-stressing tasks are dangerous and can cause pain") and the behavioural effects ("avoidance of back-stressing tasks generally reduces pain") based on the generalization "Since back-stressing tasks can cause pain, and avoidance of these tasks generally reduces pain, it is best to avoid such tasks" (Goubert et al. 2011).

According to TT, the mental states referred to in FP are the states that feature in our everyday common-sense mental explanations. TT asserts the view that, as theoretical claims, pains are not directly observed, though they are assumed to explain observable effects like

pain facial expression, guarding, verbal self-report and other pain behaviours (e.g., Goubert et al. 2011). In addition, TT asserts that everyday common-sense attributes a number of features to mental states, such as causal and subjective features. For example, TT claims everyday common-sense attributes two kinds of features to pains (e.g., Butler et al. 2003; Aldrich & Eccleston, 2000; McCrystal et al. 2011; Moseley, 2007). First, there are subjective properties. Pains are subjective or personal experiences, which depend for their existence on feeling them. Since pains are known by personally feeling and coming to know them in that way, they are private. The claimed privacy of pain seems to contrast with the *objective* nature of objects of standard perception. For example, the book on the table can be seen by others in just the way I see it, so is not private or subjective like personal pain. Second, there are causal properties. Pains are the kind of experiences that are caused in specific circumstances, and which reliably produce pain behaviours. Pain is causally connected to goal-directed behaviours that are helpful. Some causal roles pain can have are:

17. Perceiving pain might cause immediate protective behaviours
18. Anticipating pain might cause avoidant and protective behaviours
19. Expressing pain might cause palliative treatment from others
20. Expressing pain might cause reassuring interpersonal interactions
21. Labelling pain might cause a person to create a meaningful personal narrative

These complex subjective and causal features of pain FP are the primary focus of pain EM which challenges the propriety and explanatory value of pains.

2.2 Elimination of Folk Psychology and Radical Theory Change

The second central argument for EM begins with the claim that FP is mistaken about the nature of the mind. EM suggests that the theoretical framework of FP is a radically false misdescription of cognition and private mental states such as pain; thus, the claims of FP designate nothing that is real (e.g., Churchland, 1981, 1996; Churchland, 1989; Dennett, 1978; Hardcastle, 1999; Roth, 2012; Stich, 1983). Although EM concerning pain agrees with the type identity theory of mind that everyday pain is nothing more than physiological mechanism (i.e., the NEIM), it asserts that pains cannot be type identified with or reduced to physiological mechanism. The reason pains are irreducible is not because they are *nonphysical*, as dualism claims; instead, it is because pains, as proposed by common-sense psychology and many philosophies of mind, do not *really* exist.

To clarify this radical claim, it may help to make a distinction between *ontologically conservative* (retentive) theory change, and *ontologically radical* (eliminative) theory change (Savitt, 1974). Ontologically conservative theory change happens when the concepts and claims of the old reduced theory are revised and relocated in the new reducing theory. The scientific wave theory of visible light, first proposed in the 1660s by Robert Hooke, was replaced roughly 230 years later by the theory of electromagnetic radiation (Baierlein, 2002). A key event in the change from the old to the new theory of visible light was the Michelson-Morley experiment (1887), which led to the eventual rejection of the concept of the luminiferous ether, the claimed medium of light transmission, while the idea of wavelength was retained (Baierlein, 2002). Moreover, at no point in the change did we come to say that visible light is not real. Instead, visible light was type identified with electromagnetic radiation.

Alternately, the scientific theory of caloric fluid was *rapidly eliminated* from scientific theories of heat (Lyons, 1985). There is nothing in the theories of static and kinetic

friction (dry friction) that we can correctly type identify with caloric. The theory of caloric fluid does not mesh with many other parts of well established physical science, and is thus far removed from anything we now claim about dry friction or electromagnetism that was once explained by caloric fluid. Accordingly, the dramatic progression from caloric fluid to modern explanations of heat was *ontologically radical*. Caloric fluid was eliminated from our ontology, and we came to understand that the concept is false: it designates nothing *real*.

EM predicts that an ontologically radical theory progression of this kind is expected of FP. Just as we came to understand that there are is no such thing as caloric fluid, so EM predicts that FP concepts such as pain will eventually be recognized as false concepts. Since there is nothing that has the subjective and causal properties we assign to pain it will likely turn out that *there really is no such thing.*

2.3 Folk Psychology and Explanatory Power

A reason to prefer type identity theory of pain compared to other philosophies of pain is that it has superior *explanatory power* compared to the alternatives. Similarly, advocates of EM have argued that any accurate theory should provide a research program that meshes with well-established science and which possesses considerable explanatory power (Churchland 1981, 1993, 1996; Churchland 1989; 2002). However, FP appears to be relatively *explanatorily powerless*, since there are many mental phenomena that FP cannot explain. For example, questions concerning memory and learning, motivation, dreams, coma, the dementias, pain disorders (e.g., CIP) are entirely overlooked by FP compared to the many neuroscientific theories based on reliable correlations between mental states and specific physiological states measured both in humans and animals. Since it is appropriate to support theories that offer the best explanations of mental phenomena in their domains, all other things being equal, then it is rational to endorse those theories, compared to the alternatives.

Since neuroscientific theories satisfy this stipulation much better than FP, EM philosophers infer that EM is explanatorily superior compared to FP (Churchland 1981, 1993, 1996; Churchland 1989; 2002).

Indeed, the record of folk theories such as folk physics, folk biology, and folk epidemiology shows that they all turned out to be *radically* wrong (Churchland, 1981; Churchland 1989). Thunder is not Zeus hurling lightning bolts; it is the sudden increase in pressure and temperature which produces rapid expansion of the air surrounding and within a bolt of lightning. Bubonic plague is not God's punishment for sin; it is a rat-borne bacterial infection. Pain is not a feeling in the nonphysical mind; it is the specific physiological operations of the NEIM. Since folk theories generally turn out to be false, it seems unlikely that FP will prove to be true. Finally, since FP concerns a subject that is far more complex than any previous folk theory; namely, intelligent human behaviour, it seems unreasonable, all other things being equal, that this one time folk theory really got something right (Churchland, 1981; Churchland 1989).

3 Pain Eliminative Materialism

Philosopher Daniel Dennett (1978) proposes that the pain FP is false and ought to be eliminated. His argument draws on reports of clinical pain conditions which he characterizes as the *reactive dissociation* (RD) of pain emotion from its somatosensory aspects. Some surgical procedures, hypnotic protocols and drugs reduce or remove the negative emotion of pain (i.e., its characteristic unpleasantness) while retaining its somatosensory-discriminative features such as pain intensity, location, and so on. These reports come from pain patients who have had prefrontal lobotomy (e.g., Bouckoms, 1994; Freeman et al. 1942; Hardy et al. 1952) or bilateral anterior cingulotomy (e.g., Foltz & White, 1962; White & Sweet, 1969; Wilkinson et al. 1999; Yen et al. 2005) as a final recourse for their debilitating chronic pain,

from patients under the effects of hypnotic suggestion (e.g., Barber, 1963; Rainville et al. 1997), NO and some opiates like morphine (Barber, 1959). These patients typically report post-treatment that they can still perceive somatosensory features of pain qualia following painful stimulation, but they no longer feel the experience to be unpleasant or aversive.

Dennett (1978) argues that the case of RD in pain patients radically falsifies pain FP. At issue is the core FP pain generalization, (1) "Pain generally hurts and is unpleasant" and the FP pain claim (1*) "Pain is always subjective and private". According to Dennett, an RD pain patient sincerely believes that (a) he is in pain, and that (b) his pain is not painful at all. Given (1) and (a), we may infer that he is in pain. But this contradicts his belief (b) that is guaranteed to be true assuming (1*). Therefore, pain FP is contradictory. Since anything with contradictory features cannot exist, it follows that nothing can be a pain; thus, pain FP designates nothing that is *real*. I will consider two objections to Dennett's pain EM.

Kaufman (1985) endorses Dennett's conclusion that nothing is picked out by pain FP, but disagrees that this justifies the *elimination* of pain FP. According to Kaufman, the correct conclusion to derive from Dennett's argument is that pain FP is simply in *error* to believe that the conjunction of (1) and (1*) is necessary. Specifically, pain FP is right to assert (1) as a true generalization, but wrong to assert (1*) as a true *claim*. Instead, Kaufman (1985) thinks they are both general statements. Thus, to avoid the threat of elimination posed by Dennett's argument, he suggests that the mistaken claim (1*) be modified and presented as a FP generalization. Replacing claim (1*) "Pain is always subjective and private" with the generalization (1**) "Pain is generally subjective and private" yields the new pain FP conjunction (1) and (1**), which is compatible with Dennett's argument (Kaufman, 1985).

Conee (1984) and Guirguis (1998) have objected that (1) and (1*) are not actual parts of pain FP. The reports of the RD pain patients do not imply that pain is not real. Rather, according to Conee (1984) and Guirguis (1998), such cases reveal the complexity and

intricacy of pain FP: what appears to be simple in everyday common-sense thinking turns out to be more complex in scientific observation. Hardcastle (1999) also thinks that the physiological basis for pain is so complex that no one thing answers to our pain FP. Although Hardcastle describes pain FP as a "myth", she does not appear to think that pain is not real, but rather that it is much more complex than pain FP appears to think it is. This objection challenges a precise reading of (1*).

The RD pain reports show that the negative emotional features of pain are not necessary for an experience to be typed as pain. Thus, the type identity of pain involves somatosensory features rather than negative emotional features (e.g., Ploner, 1999). However, instead of undermining Dennett's argument for pain EM, I think this point actually endorses its overall purpose. The purpose of Dennett's argument is not merely to show that pain FP is actually falsified by science, but also that the *limit* of what can be reduced or eliminated as a result of science is *much nearer* than we may intuitively think. Dennett's pain EM implies that, appearances to the contrary, intuitive judgments are insufficiently reliable to launch definitive predictions about whether in the future something will be reduced or eliminated (or not) as a result of science (Churchland, 1981, 1996; Churchland, 1989; Dennett, 1978).

The rest of this chapter will consider five objections to EM. I will focus the discussion on pain EM.

4 The Case against Pain Eliminativist Materialism

4.1 Pain Folk Psychology and Explanatory Power

In response to the EM criticism that FP is explanatorily powerless, many philosophers have objected that this negative evaluation is incorrect. FP has actually stimulated a number of productive research programs in scientific psychology (Greenwood, 1991; Horgan and Woodward, 1985). Pain FP has had a similar effect in pain cognitive psychology and in

clinical pain medicine, but I will show that outcomes concerning the explanatory status of FP are as yet inconclusive. The following two cases substantiate this guarded assessment.

One case concerns FP generalization (4) that pain intensity is sensitive to changes in the weather. This generalization has been intensively investigated for over five decades. However, experimental results are mixed. De Figueiredo et al. (2011) conducted a meta-analysis of 247 abstracts of studies that investigated weather-osteoarthritis pain relations and found a strong correlation between weather changes and increased pain in osteoarthritis patients, especially changes in atmospheric pressure, but not in precipitation. Ngan and Toth (2011) found that weather-mediated changes occur for patients with neuropathic pain, manifesting as relief from Chinook winds, while cold temperature conditions provoked exacerbations in neuropathic pain intensity. However, other studies reveal no pain-weather relationship (e.g., Fors et al. 2002; Smedsland & Hagen, 2011; Wilder et al. 2003). For example, weather variables have explained only a small amount of change in individual differences between patients in their weather sensitivity patterns. Gorin et al. (1999) found that while rheumatoid arthritis patients with higher levels of self-reported pain showed more weather sensitivity, weather variables accounted for only a small amount of change in pain scores. This pattern was true even for patients with the most pronounced pain-weather correlations. Hence, although weather sensitivity was shown, the effect sizes were not clinically meaningful. Similarly, Çay et al. (2011) found that the belief about the presence of weather-arthritis correlations was *stronger* than its statistical power. Thus, further study is warranted concerning the explanatory power of this FP generalization. In the meantime, caution seems to be the order of the day.

Many pain psychology studies demonstrate that pain FP beliefs and attitudes are correlated with the development of chronic pain and disability (e.g., Aldrich & Eccleston, 2000; Eccleston et al. 1997; Leeuw et al. 2008; Main et al. 2010; Moseley, 2007; Williams &

Thorn, 1989). Specific beliefs that set the stage for activity restrictions are associated with the development of chronic pain and related disability. These beliefs include the pain FP generalizations such as "hurt is generally harm" (i.e., if it hurts, something serious is broken), "pain is a signal to stop what you are doing" (i.e., if an activity results in pain, a person should always stop before an injury occurs), and "rest is the best medicine" (i.e., pain signals that a person should rest to recuperate the body). Integration of pain FP in clinical pain management and treatment has been shown to improve patient outcomes and reduce pain. For example, decreases in the belief that pain signals damage are correlated with decreases in patient disability (e.g., Elander et al. 2009; Jensen et al. 2011; Molton et al. 2009). Increases in perceived control over pain and decreases in pain catastrophizing and in the belief that one is disabled are also associated with decreases in self-reported patient disability, pain intensity, and depression (e.g., Elander et al. 2009; Jensen et al. 2001; Molton et al. 2009). Clearly, pain FP has stimulated important research in cognitive and social pain psychology which has translated into some improved patient treatment outcomes. Inquiry into pain FP appears justified for its own sake, because informed pain clinicians can find new opportunities to intervene in modifying maladaptive beliefs concerning pain. According to supporters of pain FP, the folk theories pain patients hold are real because they produce *genuine effects* on how patients act and interact with caregivers and physicians. Thus, in this area at least, the EM criticism that pain FP is explanatorily stagnant appears mistaken.

4.2 Pain Folk Psychology, Introspection and Verbal Self-Report

Even if it is granted that pain FP is theoretically incomplete, or fails to explain every feature of pain, it does not follow that it is therefore *radically* false (e.g., Horgan & Woodward, 1985). Supporters of pain FP object that the theoretical considerations offered in EM cannot falsify the evidence provided by the everyday, immediate consciousness of our

own minds and bodies, such as our *introspective experience*, which seems to intuitively compel the belief in the essential subjectivity and privacy of pain. The experience of pain in the mind appears to be a function of the features and causal impact of a noxious stimulus on a person. What happens in the mind is a *mental representation* of triggering noxious events, either external or internal (bodily or visceral). According to pain FP, the mind reflects the external or internal environment through subjective pain experience. This leads to the FP claim that verbal self-report of pain is a function of a mental representation to which the person has privileged introspective access (Aldrich & Eccleston, 2000; Butler et al. 2003; McCrystal et al. 2011; Moseley, 2007; Williams & Thorn, 1989).

The folk belief in the subjectivity and privacy of pain has direct consequences in clinical neurological practice. For example, some neurological outpatients have medically unexplained pain, which is correlated with high levels of psychiatric comorbidity (i.e., somatoform disorders) (Barksy & Borus, 1999; Kirmayer et al. 1994; Smith, 1992; Sullivan, 2001). These patients are extremely reluctant to accept mental-based (i.e., psychological) explanations for their pain because psychological symptoms are commonly believed to be shameful and closely related to the social stigma of "diseases of the mind". In contrast, "physical" pain is believed to be free of stigma or implied blame. The difficulty patients with somatoform disorders have in accepting psychological explanations for their pain symptoms partly derives from, and reinforces, FP claim (13) concerning the dualistic nature of pain as mental rather than physical. Miresco and Kirmayer (2006) found that mental health workers also used FP claim (13) to reason about patient responsibility for pain. When patient pain was believed to have a psychological etiology, the patients were more often thought to be responsible for their condition. But when the pain was believed to have a physiological cause, the patients were considered *less* blameworthy. In another survey study, Demertzi et al. (2009)

found over a third of health-care workers endorsed dualistic views on the nature of mind and body.

Some EM philosophers caution that we should be deeply suspicious about the reliability of introspective 'evidence' concerning the functions of the mind (Churchland, 1981; Churchland, 1989; Smart, 1959; Wittgenstein, 2009). If introspection is as theoretical as many philosophers and psychologists now consider *exteroception* to be, what we introspect may be mostly determined by the theoretical framework of FP. That is, words like 'seeing' pain 'in the mind' may be the same as people 'seeing' pain as demonic possession (Churchland, 1981; Churchland, 1989, 2002; Dennett, 1978; Hardcastle, 1999).

Skepticism about introspection is supported by a psychophysical study that used causal modeling analyses (i.e., path analysis) to determine the accuracy of verbal self-reported pain ratings (Chapman et al. 2002). I will now describe this study in some detail. In causal modeling, a target variable is regressed on a group of other variables that, based on the model, are causes. The aim of causal modeling is to *infer the best explanation* of a phenomenon consilient with established science. In Chapman et al. (2002), this requirement entailed consilience with established theories of the nervous, endocrine and immune systems and their functional interdependencies concerning pain. The study involved 100 subjects (56 males, 44 females). The subjects experienced three levels of noxious finger-tip electrical stimulation. On each experimental trial, Chapman et al. (2002) recorded heart rate, skin conductance response, pupil dilation, and event-related late near field evoked potentials. They also collected verbal self-reported pain ratings from the subjects following painful stimulation.

Chapman et al. (2002) used stimulus intensity level as the standard against which to assess the intensity of the verbal pain self-report. Accuracy was computed by calculating for each experimental subject the squared nonlinear correlation ratio (η^2); that is, the proportion

of variance in the pain report that the stimulus level can explain: $\eta^2 = 1{:}0 - (SS_{err}/SS_{tot})$. Cohen (1988) specifies that coefficients greater than .5 indicate moderately high levels of agreement, while coefficients greater than .7 indicate excellent agreement. Chapman et al. (2002) found that the accuracy estimated by these procedures ranged from .7 to .91 with a median of .64. Using the criterion (.5) specified by Cohen (1988), Chapman et al. (2002) reasoned that the majority of the subjects showed a moderately high level of accuracy.

Next, the authors used causal modeling to determine the accuracy of pain self-report intensity ratings. Chapman et al. (2002) used the following accuracy predictors: (1) electrical current intensity, (2) attention (event-related late near field evoked potential N150 amplitude[15]), and (3) arousal (overall sympathetic nervous system arousal based on heart rate, skin conductance response, and pupil dilation). The final causal model developed in the study showed a direct causal chain that linked electrical current intensity and accuracy. First, electrical current intensity determined attention. Second, attention in turn determined the intensity of arousal. Third, arousal proved to be the *sole determinant* of the accuracy of the verbal pain self-report. Finally, male subjects who experienced higher levels of arousal gave more accurate pain reports than those who had lower levels, but female subjects who had higher levels of arousal gave less accurate pain reports than those with lower levels. The study demonstrates construct validity[16], not from direct stimulus-response correlation, but from a *causal chain* that related stimulus to response.

So, what do the results of Chapman et al. (2002) imply for the alleged reliability of introspection and the mental representation of noxious events? If, according to pain FP, a mental representation of a noxious stimulus is what the mind accurately produces in response to a noxious trigger – whatever the representation might be – then, given Chapman et al.

[15] The N150 is a negative event-related potential (ERP) that peaks roughly around 150ms following stimulus onset.

[16] In experimental science, construct validity refers to the extent to which inferences can be made from the operationalizations in a study to the theoretical constructs on which those operationalizations were based.

(2002), it does not appear to determine the accuracy of pain verbal self-report. Given the FP claim, the nature and intensity of the alleged mental representation should determine the accuracy of subjective and private self-report of pain. However, the results from causal modeling analyses in Chapman et al. (2002) challenge the alleged necessity of this claim.

4.2.1 Pain Folk Psychology and Verbal Self-Report

Language occupies a privileged position in pain FP, yet the limitations of verbal pain self-report should be recognized. Linguistic competence slowly emerges in the course of normal development (Craig, 2006; Stanford et al. 2006; Wittgenstein, 2009). Thus, it is not fully available to all individuals; including neonates, infants and young children, individuals who do not have the language of the pain caregiver, and those individuals with acute, chronic or acquired cognitive or physical disabilities (Hadjistavropoulos et al. 2011). Even individuals with effective linguistic and social skills can find it difficult to describe personal pain. This is because pain is a complex experience that involves somatosensory, negative emotional and cognitive features. Moreover, psychological studies show that, in clinical and nonclinical settings, nonverbal pain behaviours such as facial expression are viewed as more credible by observers and more accurate of the presence of pain (e.g., McCrystal et al. 2011; Schiavenato & Craig, 2010). Pain facial expression has been reliably observed in a broad range of populations, including the special populations just mentioned (e.g., Grunau et al. 1987; Hadden et al. 2002; Kunz et al. 2007; Kunz et al. 2008; Nader et al. 2004), and in nonhuman mammals (Keating et al. 2012; Langford et al. 2010; Leach et al. 2012; Sotocinal et al. 2011). In an fMRI study, Kunz et al. (2011a) found that verbal self-report pain ratings were not related to facial expressiveness and did not differ between painful trials with and without facial expressions. The authors inferred that the variability in facial responses and the correlated neural responses could not be explained by differences in verbal pain reports. Thus,

pain facial expression appears to embody unique features of nociception within the cortical parts of the neospinothalamic tract. Given the involvement of the neospinothalamic tract in pain, as made clear in Chapter Two, the finding of Kunz et al. (2011a) indicates that facial expression reflects some features of pain not fully accounted for in verbal self-report, consistent with clinical and behavioural interpretations (Craig et al. 2011; Prkachin, 2009; Williams, 2002).Contrary to the claims of pain FP, verbal self-report is not *the* gold standard of pain.

Psychological studies show that pain self-report may confound pain experience with the need to influence those people attending to what the person says (Craig, 2009; Craig et al. 2011; Hadjistavropoulos et al. 2011; McCrystal et al. 2011; Schiavenato & Craig, 2010). Thus, speech is a function of *both* conscious experience and perceived best interests. The audience may not be disposed to care for the person in pain; people may respond by ignoring the person in pain, punishing them for the pain report, or exploiting them because they are vulnerable (e.g., Craig, 2009; Schiavenato & Craig, 2010). Some pain patients have skill in negotiating the social complexities of health care settings, whereas others require devoted clinical attention (e.g., Elander et al. 2009; Jensen et al. 2001). Importantly, these responses and contextual demands that influence verbal self-report operate *outside* conscious introspection; that is, they are type identical to the operation of *unconscious neurophysiological mechanisms* (Churchland, 1981; Churchland, 1989, 2002, 2011; Craig et al. 2011; Dennett, 1978; Hadjistavropoulos et al. 2011; McCrystal et al. 2011).

4.3 Pain Eliminativist Materialism is Self-Refuting

Some philosophers have argued that EM is self-refuting (e.g., Baker, 1987; Boghossian, 1990, 1991; Reppert, 1992). The objection is that a capacity or activity that is somehow invoked by EM is *itself* something that requires beliefs. A reasonable choice for

this activity is making an assertion. Plausibly, to assert something one must *believe* it (Baker, 1987; Boghossian, 1990, 1991; Reppert, 1992). Thus, for EM to be asserted as a philosophy, the advocate of EM must believe that it is true. However, if the advocate of EM believes this, then there are beliefs and EM is therefore false. Similarly, to assert that there are no pains, the EM philosopher must believe that this assertion is true. If this is so, then there are beliefs, and EM is proven false.

In response to this objection, EM philosophers have observed that the mere idea that there are no pains is not *itself* contradictory (Churchland, 1981; Churchland, 1989; Ramsey, 1991). Thus, the objection is not that EM is self-refuting. Instead, it is that the EM *philosopher* is doing something that refutes his or her own philosophy. In the previous example, the refuting act is making an assertion, as it is declared by the supporter of FP that we must believe anything we assert. However, this last claim is exactly the type of claim FP makes that EM is urging we should reject. According to EM, all of the diverse capacities that we now explain by appealing to beliefs do not *really* involve beliefs at all. All of the capacities we currently account for by invoking pains do not really involve pains. Thus, the self-refutation objection begs the question against EM.

4.4 Pain Eliminativist Materialism is a Premature Philosophy

Some philosophers claim that EM is premature, given its apparent promissory nature (e.g., Baker, 1987; Fodor, 1974; 1987; Greenwood, 1991; Horgan & Woodward, 1985). EM claims that the correct theory of pain, when discovered by neuroscientists, will not reveal anything like the pains FP describes. Hence, for EM to work, an assumption needs to be made that neuroscience is going to turn out a specific way. But, why make that assumption before science gets there? Why infer an ontologically eliminative conclusion concerning pain, when a core premise required for that conclusion is unknown?

To the charge that EM is promissory in nature, EM philosophers have responded that this misconceives the nature of EM (Churchland, 1981, 1996; Churchland, 1989). EM is eliminative in *predicting* the future elimination of folk psychological pain from our post-neuroscientific ontology. Just as oxidative reactions as described within elemental chemistry bore no resemblance to phlogiston release, or dry friction to caloric fluid, continuing development in neuroscience will *likely* reveal that there are no such things as pains as understood by everyday common-sense. This is not predetermination in action: rather, it is theoretical expectation based on the historical and present success of science. Still, this prediction might prove to be quite wrong. Thus, EM urges we adopt a wait-and-see attitude.

A supporter of FP might respond that in making this point concerning empirical likelihood, the EM philosopher is now making an empirical prediction, thus confounding EM as a *metaphysical theory of mind* with EM as a *scientific theory*. Is pain EM a philosophy of pain, or a science of pain? Defenders of EM have replied that this question is confused since the approach adopted by EM is consilient with the philosophical tradition that is as old as Aristotle and John Locke; it is naturalistic and pragmatic, as opposed to supernaturalistic or a priori (see Table 3, 'Metaphysical', 'Theoretical') (Churchland, 1981; Churchland, 1989; 2002, 2011). Clearly, EM is thought to be part of *naturalism* (Papineau, 2002, 2009). The point was made in Chapter One, but it is worth remembering that most physicalists support methodological naturalism as a matter of fact and increasingly use the same methods of investigation as the natural sciences (e.g., Abraham et al. 2008; Churchland, 2011; Grant & Liu, 2012; Kavanagh et al. 2011; Watson et al. 2012). According to EM – and the type identity theory of pain offered in this thesis – metaphysics is *continuous* with the sciences and not separate from them.

Another EM response to this objection is to consider the *broader theoretical place* EM can have in the development of a successful theory of pain or the mind (Churchland,

1981; Churchland, 2002). Many philosophers have stipulated necessary conditions that any theory of the mind must satisfy. Some of these philosophies require that the conditions include the explanation of mental states as characterised by FP. Thus, if a theory doesn't include pain, then it isn't a complete account of *real* pain (e.g., Baker, 1987; Chalmers, 1996; Fodor, 1987; Greenwood, 1991; Horgan & Woodward, 1985; Searle 1992; Tye, 2006). An advantage of EM is that it releases philosophical theorizing from this framework (Churchland, 1981; Churchland, 1989). This means that the nature of the relationship between EM and science, similar to the relationship between type identity theory and science, is actually more interdependent and intimate than many philosophers have assumed. That is, EM, like type identity theory, is heavily reliant on the development of a scientific account of mind. Unlike type identity theory, however, EM appears exclusively reliant on the development of an *ontologically radical* scientific account of mind (Savitt, 1974). Notwithstanding comparisons between type identity theory and EM, the immediate point is that radical philosophizing about pain or mental states generally may itself rest upon our acknowledging the *likelihood* that our common sense point of view may be deeply wrong.

4.5 The Success of Pain Folk Psychology

The final objection I will consider of pain EM, which develops the criticism described in section 4.1, is that it ignores the enduring *success* of pain FP, success that shows it offers a more accurate account of pain than EM can fathom. Human beings are very successful in using everyday common-sense to predict the pain and pain-related behaviours of other people. For example, a person can form a maladaptive cluster of behaviours around pain FP (Aldrich & Eccleston, 2000; Butler et al. 2003; Moseley, 2007; Williams & Thorn, 1989). Chronic pain patients may experience pain with no identifiable pathology, yet they may *avoid* beneficial activities because they fear that physical harm will occur. Pain clinicians are

trained to help patients identify and reform this theoretical link by (e.g.) teaching about different kinds of pain (e.g., acute versus chronic pain) and how pain may not necessarily signal bodily harm. Such education has been successful in replacing maladaptive pain folk generalizations and claims with adaptive folk generalizations and claims (e.g., Elander et al. 2009; Jensen et al. 2011; Molton et al. 2009). Kitcher (1984) and Fodor (1987), both supporters of FP, have observed that the success of FP is something like an inference-to-the-best-explanation argument in favour of FP and against EM. The best explanation for the success in explaining and predicting human and animal pain behaviour, and in reforming maladaptive pain FP, is that pain FP is *approximately true*, and that there really are pains.

However, one EM response to this argument is to re-assert that *any* theory can *seem* successful even when it is completely false (Churchland, 1981; Churchland, 1989, 2002). Our intellectual history shows that human beings often discredit deviations and inconsistencies, ignore failures as unimportant, and typically assign more success to a well-known theory than it warrants (e.g., Gribbin, 2004). Like the supporters of caloric fluid or luminiferous ether, we may be blind to the errors of pain FP until an alternative (neuroscientific) explanation is available (Churchland, 1981; Churchland, 1989, 2002). Since neuroscience is still a young science, we likely have only a faint understanding of what remains to be discovered about pain, and only a faint understanding how the discoveries will alter our pain FP. Although pain FP currently shows many functions beyond explaining and predicting, that fact doesn't change its *theoretical status* nor protect its generalizations and claims from elimination (Churchland, 1981, 1993, 1996; Dennett, 1978).

It appears that the success argument for the approximate truth of FP is a case of fallacious reasoning called the *base rate fallacy* (cf. Howson, 2000; Lipton 2004). To illustrate this fallacy, consider the following. There is a test for a disease for which the rate of false positives (positive results in instances where the disease is absent) is one in ten (i.e.,

disease-free persons test positive 10% of the time), and the rate of false negatives (negative results in instances where the disease is present) is zero. Now, if a person tests positive, what is the likelihood that a person has the disease? Based on the rate of false positives, it would be an error to infer that the likelihood is 90%, since the *actual* likelihood relies on the base rate of the disease in the population (i.e., the proportion of people having it). This means that the lower the base incidence of the disease in the population, the lower the likelihood that a positive result shows the presence of the disease. Similarly, claiming the success of FP as an indicator of its approximate truth (assuming a low rate of false positives) is arguably a case of the base rate fallacy. The success of a theory does not by itself indicate that it is likely approximately true. Since there is no *independent* way of knowing the base rate of approximately true theories, the likelihood of pain FP being approximately true cannot be determined.

Finally, pain FP is just like any theory in that it can be partly right and partly wrong. The history of science reveals many instances where the generalizations and claims of a mistaken theory are neither smoothly reinstated in a new theory, nor completely eliminated (Baars, 2012; Churchland, 1981; Churchland, 1989, 2002). Instead, it is substantially altered, with perhaps only some of its generalizations and claims being fully eliminated. The altered conception of pain that is used in pain neurophysiology education (PNE) is a case in point. According to PNE, pain is defined as being identical to complex neural processing and adaptation rather than the traditional theory of spinal pathology (Moseley, 2003, 2005; Moseley et al. 2004). Studies have shown that PNE leads to some normalization of attitudes and beliefs about pain, a reduction in pain catastrophizing (Sullivan et al. 1995), and an improvement in physical performance (Clark et al. 2011). With regard to the pain FP generalizations and claims (1)-(21) cited above, following a single or multiple PNE sessions, pain patients are less likely to believe pain is indicative of tissue damage (5); less likely to

seek care from others when in pain (7), more likely to believe one can control one's pain (10), and perceived themselves as less disabled (e.g., Moseley, 2003, 2005; Moseley et al. 2004). Thus, EM may be viewed as an end-point on a continuum with *many* possibilities falling somewhere in between. One possibility is that pain FP will only be eliminated to a certain extent, and that various aspects of pain FP will be at least partly confirmed or retained. Another possibility is that *neuroscience* theories of pain will face falsification or elimination and new neuroscience theories will become integrated with pain FP, a possibility which actually occurs in PNE. A type identity theory of pain might be accommodated here. Nonetheless, as EM correctly observes, considerations of theoretical accommodation do not determine the *ontological status* of a theory, nor inoculate its claims and generalizations from elimination.

4.6 Theory-Theory and Simulation Theory

As described above, a main argument for EM is the idea of theory-theory (TT). TT claims we use a theoretical framework to explain and predict human behaviour, and to assign mental states to others. A critic of TT, *simulation theory* (ST), challenges the importance of theorizing in FP, and instead proposes that FP is a body-based simulation, and bodily states such as pain are a foundation of intersubjective phenomena such as pain empathy (e.g., Avenanti et al. 2005; Engen & Singer, 2012; Gallese & Goldman, 1998; Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007; Rizzolatti & Arbib, 1998; Rizzolatti et al. 2001). ST claims that it is the *embodied imitation* of the observed body in pain that enables us to recognize others as persons in pain, not an abstract, theory-based inference.

The finding of mirror neurons in humans and macaque monkeys has been used in support of ST. Mirror neurons are a subset of neurons in the frontal cortex of the monkey that respond both when the monkey performs a type of action and when it observes other

individuals perform actions of the same type (e.g., Iacoboni & Dapretto, 2006; Kohler et al. 2002; Pellegrino et al. 1992; Rizzolatti & Craighero, 2004; Rizzolatti et al. 1996; Rizzolatti et al. 2001). In a classic study of F5 neurons in the macaque monkey, Rizzolatti et al. (1996) found that roughly 60% of the F5 neurons recorded from were specific for one type of action (e.g., grasping). Some were *highly* specific, selectively firing during the observation of a specific type of hand configuration used to grasp or manipulate an object (e.g., a precision grip, but not whole hand prehension). The remaining neurons were activated by the observation of two or more hand actions. The actions most represented were: grasp, manipulate, and put object on a surface in front of the monkey. For example, during one single-cell recording reported in Rizzolatti et al. (1996), the F5 neuron recorded from discharged when the experimenter rotated his hands in opposite directions around a small piece of food; but not during the observation of grasping actions. However, when the monkey made wrist rotations to take away the food from the experimenter's hand, the neuron responded. Meaningless actions, actions without an object, the simple presentation of an object, or action by a hand where the rest of the body could not be observed, did not fire these specific neurons in the monkey (Rizzolatti et al. 1996). Thus, it appears that the relationship between specific *hand* action types (e.g., precision grip) in observation/performance contexts, and operations of specific cell types, is one of *type identity*. Although these data are relevant to this project because they count as evidence of type identity, the philosophy of ST does not entail type identity theory of mind, and ST theorists have not endorsed it in their writings.

Indirect evidence in support of a mirror neuron system (MNS) in humans is partly based on the reactivity of cerebral mu rhythms during action observation (e.g., Cochin et al. 1998; Gastaut & Bert, 1954; Oberman et al. 2005; Pellegrino et al. 1992). Mu suppression and enhancement describe sensorimotor operations in frontoparietal cortical networks. Electrophysiological studies using EEG and MEG demonstrate that observing an action

performed by another individual suppresses the mu rhythm of the observer, thus providing evidence for a *cortical resonance mechanism*, which connects the observed action to the action of the subject's own motor repertoire (e.g., Cochin et al. 1998; Gastaut & Bert, 1954; Oberman et al. 2005; Pellegrino et al. 1992). In electrophysiological studies of pain empathy, Yang et al. (2009) found that females showed stronger mu suppressions than males when observing painful and non-painful stimuli, and only females showed a positive correlation between mu suppression for pain empathy and the personal distress subscale of the Interpersonal Reactivity Index, a measure of empathy. In Perry et al. (2010), subjects were told to imagine the feeling of the person whose hand was shown and to assess his or her state of emotion. The authors found that the pain condition stimulated greater mu suppression than the no-pain condition over frontoparietal cortex. There was also a significant interaction between pain and similarity for similar others (i.e., the hand shown of a normal adult), but in the dissimilar-other group (i.e., the hand shown of a patient with a neurological disease), suppression was equally large in the pain and no-pain conditions. Perry et al. (2010) inferred that mu suppression is evoked involuntarily in pain empathy, even if the other person is different from oneself. These findings support the claim of ST that the experience of pain empathy consists in involuntary sensorimotor resonance between self and other, according to which the observer both simulates and shares the pain of another person. This means that when we observe a person in pain the very same mechanisms that control the personal experience of pain may become active in the observer's frontoparietal cortex.

The results of several neuroimaging studies in special populations appear to challenge the philosophy of ST. For example, Danziger et al. (2008) tested ST using fMRI in a study of patients with CIP. The authors of the study reasoned that CIP patients cannot rely on cortical resonance mechanisms to understand the pain of others, since they are unable to personally experience pain. However, in both CIP subjects and in normal control subjects, Danziger et al.

(2008) found similar hemodynamic responses to observed pain in ACC and AINS. In another fMRI study, Aziz-Zadeh et al. (2012) found that when individuals view others experiencing pain in body parts that they themselves have, the AINS and S1 and S2 were activated, in broad agreement with ST and the cortical parts of the claimed resonance mechanism. However, when an individual views others experiencing pain in body parts that he or she does not have (e.g., a pain observer is a congenital amputee born without limbs), the AINS and S2 were *also* active, but the S1 was not. Strictly speaking, these findings do not disprove ST, since the idea of a resonance mechanism in pain empathy does not necessarily rule out the possibility that other types of responses (e.g., the stress response) or mechanisms in other physiological systems could modulate this function (e.g., Iannetti & Mouraux, 2010; van Rysewyk, 2009; Yamada & Decety, 2009). For example, in an empathy study using a modified Trier Social Stress Test, Buchannon et al. (2012) found that CORT responses were matched between speakers and observers, and were correlated with trait empathy but not with the speaker's fear or distress. This study indicates that we can 'catch' another individual's stress.

While experimental findings of cells with 'mirror' properties point tantalizingly to a MNS in humans, and support the accuracy of ST compared to TT (and possibly type identity theory of mind), more research on the hypothesized resonance mechanism using different methods and animal species is required to explain its fundamental nature and involvement in intersubjective phenomena such as pain empathy. These findings will enable more fine-grained philosophical assessment of the arguments for and against ST and TT.

5 Conclusions

Eliminative materialism concurs with the type identity theory of mind that there is nothing more to pain than physiological mechanism, and that metaphysical claims about

mind are heavily reliant on the development of a scientific account of pain. However, unlike the identity theory, eliminative materialism rejects that pain can be type identified with mechanism. This is because alternative philosophical and common-sense theories of pain are false and have no home in an accurate neuroscientific successor pain theory. Although pain folk psychology has stimulated a number of research programs in psychology which have led to improved pain patient outcomes, our own intellectual history shows that any theory can appear successful or beneficial even when it is completely false. Appearances notwithstanding, neuroscience already shows that many core folk pain claims are mistaken, while the truth of many others is not currently known. Eliminative materialism implies the unsettling consequence that the limit of what can be scientifically eliminated or reduced is much closer than we conventionally think. Despite this bleak outlook for a type identity theory of pain and folk psychology, intellectual history also offers encouragement. Sometimes a mistaken theory may not be entirely eliminated, and can survive in modified form in a new theory. Thus, radical theoretical elimination can be just one end-point on a continuum among many theoretical possibilities. Partial elimination and pain folk psychology can co-exist. Still, theoretical accommodation cannot alter the realization that all human knowledge is speculative and temporary. This insight fosters humility about the ultimate truth of our current pain claims and beliefs, both scientific and folk, and this includes ontological claims of type identity theory of mind, while it encourages a modest optimism about our prospects in the centuries to come.

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